

ARCHIVES OF OTOTOLOGY.

THE MEMBRANA TECTORIA AND THE THEORY OF TONE PERCEPTION.

(FROM THE HULL LABORATORY OF ANATOMY, UNIVERSITY OF
CHICAGO.)

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(With four illustrations on Text-Plate XIV.)

IN an article published in 1907¹ I called attention to what appeared to me to be fundamental objections to the theory that the membrana basilaris could be considered a vibrating structure. In this paper I presented arguments supporting the view that the membrana tectoria was the logical structure for stimulating the hair cells of the organ of Corti by responding in its various parts to impulses in the endolymph produced by the several tones. I shall aim here to present some additional facts in support of this theory.

We find in the labyrinth of the ear three distinct types of end organs, the organ of Corti in the cochlea, the macula acustica in the utricle and the saccule, and the crista acustica ampullaris in the semi-circular canals. These three types of end organs have a common origin in the primitive otic vesicle which is derived from invagination of the ectoderm. We may also assume that these three types of end organs, however they may differ

¹ "A Restudy of the Minute Anatomy of Structures in the Cochlea, with Conclusions Bearing on the Solution of the Problem of Tone Perception." *American Journal of Anatomy*, vol. vii., No. 2, pp. 245-257.

in structure and in function, have all had a common origin in a primitive end organ, which perhaps subserved in a rude way the several functions now performed by all three. This view is borne out by an examination of the three end organs as they now occur. They still preserve certain characteristics in common which point clearly to their having had a common origin. They have in the first place, a neuro-epithelium consisting of hair-bearing cells.

In the second place, each end organ has superimposed upon the hair cells a peculiar structure, which is of epithelial origin, and against the under surface of which in each instance the hairs of the hair cells project. In the organ of Corti this superimposed structure is the *membrana tectoria*. In the *macula acustica* it is the otolith membrane, and in the *crista acustica ampullaris* it is the cupula. This analogy can safely be carried one step farther and applied to the manner in which the hair cells of the several end organs receive stimulation. It is clearly evident that in both the *macula acustica* and in the *crista acustica* the stimulation of the hair cells is brought about by movements which the otolith membrane and the cupula, respectively, receive from impulses in the endolymph. It is but rational to assume that the stimulation of the hair cells of the organ of Corti would be accomplished by the movements in its superimposed epithelial structure, the *membrana tectoria*, these movements being derived from impulses passing through the cochlea. In other words, it is hardly rational to presume that the end organ in the cochlea would, in the process of its evolution from the primitive type, change completely its manner of receiving stimuli, placing the active agent in the basilar membrane rather than in the *membrana tectoria*. Such a change seems all the more improbable since the *membrana basilaris* is a connective-tissue structure of mesoblastic origin. One would naturally suppose that in a special sense-organ, like the cochlea, the struc-

ture which takes the important rôle of stimulating the hair cells would be a structure of epithelial origin.

A study of the membrana basilaris discloses a number of conditions which render it incapable of performing the rôle of a vibrating mechanism attributed to it by Helmholtz, and also by those who have attempted to substitute other theories for the Helmholtz theory of tone perception. In the first place, I have found that, as the lower end of the basal coil is approached, the membrana basilaris quite frequently disappears at a point where a perfectly formed organ of Corti is still found. Examples of this condition are found in Figs. 1, 2, which are taken from the labyrinth of the pig. In the second place, if the radiating fibres acted, as they were supposed to do by Helmholtz, as string resonators, then we would expect to find that as the lower end of the basal coil was approached, where these fibres become shorter, they would also become thinner and more tense in order to respond to tones higher and higher in the tone scale. As a matter of fact, just the opposite condition is found, for, instead of becoming thinner as their length diminishes, the fibres of the basilar membrane become thicker and thicker, and usually lose all resemblance to a string vibrator. See Figs. 1, 3. In the third place, there exists, as a rule, directly under the tunnel of Corti, a spiral blood-vessel, which, in the labyrinth of the pig, the sheep, and the calf, I found communicated by an occasional radiating twig with the blood-vessels in the ligamentum spirale. Blood-vessels, wherever found in the body, possess the faculty of contracting or dilating, depending on vasomotor conditions and upon the blood pressure. Now, according to the Helmholtz theory, each radiating fibre or group of fibres of the membrana basilaris has its own specific energy—that is, it can respond only to tones of a certain pitch. In this way only can we account for our ability to recognize pitch. It is quite clear that, with blood-vessels

attached to the basilar membrane, the contraction or dilatation of these vessels must alter the vibration of the membrane, so that the same fibre would respond to tones of different pitch, depending on the state of the blood pressure, etc. Such a response must lead to great confusion in the recognition of pitch, a condition, however, which does not occur. This vessel under the tunnel of Corti usually exists as a small vessel, but that it may occur as a large vessel is shown in Fig. 4.

Again, it has been repeatedly pointed out that the number of radiating fibres in the membrana basilaris is inadequate to account for all the tone differences that the ear is capable of perceiving. The attempt to account for this defect in the Helmholtz theory by attributing to the ligamentum spirale the function of putting the basilar on tension, either by the filling of the numerous blood-vessels in this part, or otherwise, palpably destroys the fundamental principle of this theory, which assumes that each radiating fibre or group of fibres has its own specific energy, which makes it possible for it to respond to the impulses of but one particular tone. If we imagine that by a varying degree of tension each fibre of the basilar membrane is capable of responding to several different tones, then it is quite clear that one of the fundamental facts of tone perception would be impossible—that of recognizing absolute pitch. There is still a more serious objection to the hypothesis that each radiating fibre or group of fibres can respond to a number of different tones, depending on a varying degree of tension of these fibres. According to this hypothesis, we must assume the impossible situation that each group of radiating fibres lies in readiness to respond to this or that tone, and that, by some unknown mechanism, information of the approaching impulses of a particular tone precede these impulses and attune the fibres to receive them.

An examination of the membrana tectoria, on the other hand, shows this structure to be admirably suited

for responding to the most delicate impulses passing through the endolymph. As I have pointed out before (*l. c.*), this membrane is a delicate, semi-gelatinous structure, with a specific gravity approximately that of the endolymph. That it has a certain degree of elasticity has been shown by the work of Hardesty.¹ It is a structure that must readily respond to the most delicate impulses passing through the endolymph. From these facts the following conclusions seem justified.

First, that the membrana basilaris is not the logical structure for stimulating the hair cells, and that it is anatomically incapable of performing such a function.

Second, that the membrana tectoria is the logical structure for applying stimulation to the hair cells, and that, furthermore, it is found to be anatomically admirably suited for responding to the most delicate impulses in the endolymph.

Just how the membrana tectoria responds to the impulses produced by the various tones is another question, and one not so easy to determine, since it is impossible to construct a substance with the physical properties of this delicate membrane in order to demonstrate its mode of responding to tone impulses. The response which the membrana tectoria gives to the impulses in the endolymph must be such as to account for the phenomena associated with tone perception. The most important of these is that of tone analysis, the ability which the ear possesses of analyzing into its component parts the complex impulses which impinge on the organ of hearing, when, for example, several tuning-forks are sounded at the same time and held before the ear. Then, too, the occurrence of tone islands, or of circumscribed defects in the middle of the tone scale, must also be accounted for. It is now generally conceded that the phenomenon of tone analysis,

¹ "On the Nature of the Tectorial Membrane and its Probable Rôle in the Anatomy of Hearing." *The American Journal of Anatomy*, vol. viii., No. 2, July, 1908.

as well as the occurrence of defects in the middle of the tone scale, are localized in the peripheral apparatus, and not centrally. Both the complex character of the end apparatus in the cochlea, and the peculiar secondary phenomena associated with tone perception, argue in favor of such a conclusion.¹ Moreover, tone islands and defects in the middle of the tone scale are known to occur only in connection with diseases involving the labyrinth.²

Helmholtz was the first to point out that the various secondary phenomena associated with tone perception find their most plausible explanation in the assumption that there exists in the organ of hearing a mechanism which takes the part of physical resonator, responding in one part of the cochlea to tones of a certain pitch and in

¹ McKendrick, *Schaeffer's Physiology*, page 1165, states that a peripheral tone analysis by means of physical resonators in the cochlea "seems *a priori* to be probable, for the following reasons: (1) The existence of such (resonating) bodies would give a natural explanation of many, if not all the phenomena (associated with tone perception); (2) the evidence of comparative physiology points to a gradually increasing complexity in the structure of all terminal organs of special sense, as if there arose a necessity for differentiation and discrimination in the effects of various kinds of stimuli; and (3) investigation into the action of all the sense organs, such as those of touch and temperature in the skin, of light and color in the retina, of taste in the tongue, and of smell in the olfactory region—all indicate specialization of function in the peripheral apparatus."

On page 1192 the same author says: "The most obvious objection to any theory which dispenses with peripheral analysis is that it leaves the exceedingly elaborate structure of the organ of Corti and, indeed, of the cochlea as a whole, out of account; or, to put the matter in another light, it assigns to that organ a comparatively simple function (like that of a vibrating membrane), and one which could be performed by a more simple structure. Furthermore, the holder of such a theory, while recognizing the analytic powers we undoubtedly possess, must refer these powers to the cortex cerebri, and practically admit that the problem cannot be solved."

² For a fuller discussion of the occurrence of tone islands, see "Demonstration einer Kontinuierlichen Tonreihe zum Nachweis von Gehördefekten," by Bezold, *Zeitschr. f. Psychologie und Physiologie der Sinnesorgane*, Bd. xiii., 1896; also Bezold's *Text-Book of Otology*, English edition, 1908, pp. 53-55.

another part to tones of a different pitch. The elaboration of this theory by Helmholtz and his followers still proves to be the most satisfactory explanation of these phenomena. Helmholtz first selected the rods of Corti as the resonators in the cochlea, but later gave this up and fixed upon the radiating fibres of the membrana basilaris as the structure which should respond in the various parts of the cochlea to the tones of different pitch, and by vibrating bring about a stimulation of the superimposed hair cells. From the facts here presented, the conclusion now seems justified that it is not the membrana basilaris, but the membrana tectoria that fills this rôle of stimulating the hair cells by responding to the impulses passing through the endolymph.

In order to understand just how this membrane responds to the various tones, we must first examine its physical characteristics, since this response is a physical one. The most striking character possessed by the membrana tectoria, apart from its delicate semi-gelatinous structure, is its great variation in size from one end of the cochlea to the other. As I have already pointed out (*l.c.*), from a tiny tuft at the beginning of the basal coil, measuring in the labyrinth of the pig not over 38 micra broad, it gradually increases in size, until near the apex of the cochlea it measures fully 432 micra broad. This enormous variation in the size of the membrana tectoria is undoubtedly an important factor in determining its mode of responding to the impulses coming from the various tones. My first impression, when I became convinced that the important function of stimulating the hair cells resided in the membrana tectoria, was that the tiny tectorial membrane found at the beginning of the basal coil would be capable of responding to the impulses produced by the high-pitched tones, whereas these same impulses would be inadequate to produce movements in the much bulkier membrane found in the upper coils of the cochlea. Each succeeding tone lower in the scale might

then be capable of producing responses in a larger and larger area of this membrane. The deepest tones that the ear is capable of perceiving would in this way produce stimulations of the tectorial membrane from one end of the cochlea to the other. Such an explanation of the action of the membrana tectoria would, it seems, readily account for the phenomena of tone analysis, since for each tone there would be stimulated a different group of hair cells, and the complex of nerve impulses which reaches the centre in the cortex cerebri would be different for every tone or part of tone. This explanation of the manner of action of the tectorial membrane fails, however, to account for the occurrence of tone islands, or of defects in the midst of the tone scale. Such defects, as is well known, occur over the lower end of the tone scale as the result of obstruction in the sound-conducting mechanism. They also occur at the upper end of the tone scale, and in the midst of the scale, where they are unquestionably due to pathological changes in the cochlea itself. This well-known phenomenon has been satisfactorily explained only on the assumption that the perception of the various tones takes place in separate and distinct parts of the cochlea. Circumscribed groups of hair cells, located in this or that part of the cochlear tube, when stimulated, would result in the perception of this or that particular tone. A stimulation of circumscribed groups of hair cells in the various parts of the cochlea can only be accomplished by assuming that the membrana tectoria responds in its various parts to tones of different pitch. The tiny tectorial membrane in the lower part of the basal coil would in this way respond to the vibrations of the high-pitched tones, while the tones lower in the scale would produce vibrations in areas of the tectorial membrane in the upper coils, where this membrane has been shown to be much larger. The nerve impulses from all of the hair cells stimulated by a particular tone come together in the auditory centre in the

cortex cerebri, where the tone picture forms the final step in the perception of this tone.¹

There is, of course, still one step lacking, and that is an absolute demonstration of the action of the membrana tectoria by the construction of a model which should reproduce the characteristics of this membrane. Such a demonstration, however, owing to the extremely delicate and complex character of the membrana tectoria, does not seem feasible. On the other hand, one is not justified in arguing that the tectorial membrane cannot act in the manner which logically we are led to believe it does, simply because the physicist may not be able to demonstrate what the action of such a membrane would be. Such an argument would be the same as to deny the possibility of a photo-chemical action occurring in the retinal structures in color vision on the ground that the chemist has never been able to demonstrate such action in the laboratory.

Since publishing my first paper on the subject of tone perception, there has appeared an article elaborating the same subject by Hardesty (*l. c.*). In this paper, page 132, Hardesty makes the assertion that my theory of tone perception is based on the assumption that the membrana tectoria is constructed by lamellæ, and on page 161 he states that some of his observations "are not in accord with the premises from which my theory follows." These are the questions of the relation existing between the hairs of the hair cells and the under surface of the membrana tectoria, and the question whether the

¹ As I have previously pointed out (*l. c.*), the necessary overlapping of the areas in the tectorial membrane stimulated by two tones close to each other in the tone scale does not conflict with this theory of tone analysis, since the nerve complex which reaches the cortex must differ for the two tones, however close they may be to each other in the scale. On the other hand, this overlapping of the areas stimulated by tones near each other in the tone scale has been shown by Helmholtz to offer the most plausible explanation of the secondary phenomena of tone perception—for example, the phenomena of beats.

striation seen in a cross-section of the tectorial membrane indicates the presence of fibrillæ or lamellæ. In reply it seems almost superfluous for me to state that, in the first place, my theory of tone perception is not based on the existence of lamellæ in the membrana tectoria, and in the second place, my conclusion as to the probable action of the membrana tectoria in the function of tone analysis is that this membrane responds in its various parts to tones of different pitch. Such an action need not be influenced in the least by the attachment to its under surface of the hairs of the hair cells, or by the substitution of fibrillæ for lamellæ in its construction. Hardesty expressed in this paper, in all essential points, practically the same conclusions contained in my paper. These are: first, that the membrana basilaris is not the logical structure for responding to the impulses in the endolymph, and that anatomically it is not suited to fill such a rôle; second, that the membrana tectoria is the logical structure for carrying out this function, for which it is admirably suited anatomically; third, that the variation in the size of the membrana tectoria from one end of the cochlea to the other is the physical basis which makes it possible for this structure to perform the function of tone analysis by responding in its various parts to tones of different pitch. In his discussion of how the membrana tectoria responds to impulses passing through the endolymph, Hardesty seems more or less undecided. In one place, for example, pages 167 and 168, he argues that high-pitched tones will stimulate only the small tectorial membrane found in the basal coil, while the lowest tones would produce undulations in the entire extent of this membrane. In another place, pages 165 and 166, he accepts the conclusion which I expressed in my paper, that the tectorial membrane takes the part of physical resonator by responding in its various parts to tones of different pitch, depending on the size of the membrane. "It is probable," he states (p. 165), "that no portion of the tectorial

membrane, when subjected to sound waves transferred to the endolymph, will undergo vibrations of sufficient excursion to impinge upon the auditory hairs, except those portions whose natural periods correspond to the vibration frequency of the waves affecting the endolymph." Hardesty refers to this conception of the theory of tone perception as a modification of the telephone theory. I am of the impression, however, that Hardesty's views are after all, in the essentials, in complete accord with my own, namely, that there is a peripheral tone analysis accomplished by the membrana tectoria. This is clearly implied from his explanation of tone islands, page 172. He contends that a localized (circumscribed) calcareous deposit in the membrana tectoria would result in producing a tone island. What he evidently meant was that such a deposit would produce a circumscribed defect, not an island, in the midst of the tone scale. Now, of course, a circumscribed interference with the action of the tectorial membrane, such as a calcareous deposit of this sort would occasion, could not produce a circumscribed defect in the tone scale, unless the converse were also true, viz., that the perception of this particular part of the tone scale would result from the stimulation of that part of the tectorial membrane the movement of which was interfered with by the calcareous deposit. This brings us back again to the conclusion expressed in my first paper, that the membrana tectoria responds in its several parts to tones of different pitch, in this way filling the rôle of a physical resonator.

EXPLANATION OF FIGURES.

FIGS. 1, 2, 3.—Organ of Corti from the labyrinth of new-born pigs. Sections from near the lower end of the basal coil, showing an absence of a vibrating membrana basilaris. Ocular 4. Leitz $\frac{1}{4}$ objective.

FIG. 4.—Section same as above, but with a large vessel attached to the under surface of the basilar membrane. Also an anomaly in the rods of Corti.

A CASE OF CEREBELLAR TUMOR INVOLVING THE AUDITORY NERVE.

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MY object in reporting this case is to show the value of a very careful testing of the ear in those cases of cerebral tumor in which there are no definite localizing symptoms.

On June 15th, Dr. Carmalt Jones kindly asked me to see T. B., who had been attending his Out-Patient Department at the Seamen's Hospital, and who showed the general pressure symptoms of an intracranial tumor without any very definite localizing symptoms (*i. e.* headache, vomiting, and optic neuritis).

On Examination: General Symptoms.—There had been transient neuralgia of the right side of the face and the corneal reflex on that side was diminished, indicating slight anæsthesia of the right fifth nerve. There was ataxia of the sensory type in both hands, and on standing up with heels together and eyes shut the patient exhibited a tendency to fall to the right side; this tendency was also present when the patient was walking about. Nystagmus was not present, but developed subsequently, the movements being "slow to right side with fine movements to left." No facial paralysis.

Special Symptoms.—The patient was a boiler-maker and complained of deafness in his right ear of seven years' duration; the onset had been gradual and the deafness had been

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slowly progressing. Tinnitus was present and was of a humming character and, though varying in intensity at times, was always present, its onset being synonymous with the deafness. There was no history of pain or discharge.

On Examination.—Both membranes were slightly indrawn, the cone of light being broken on both sides. The membranes and mallei moved normally with Siegle's speculum.

Nose and Naso-Pharynx.—Nil.

Eustachian Tubes.—Quite normal when tested with catheter.

RIGHT.	TESTS.	LEFT.
On Mastoid	Weber.	Deflected to left.
Raised 2 inches	Acoumeter.	Over 12 feet.
ordinary	Voice.	
Negative.	Whisper.	4 inches.
Negative.	Rinne C.	Positive.
- 25 Seconds	Rinne C ² .	
Positive.	C. Mastoid.	22 seconds.
	Galton W.	
	Gellé.	
AIR COND ^N .		
	3 C 16	Diminished.
	2 C 32	
	1 C 64	
	C 128	- 28 seconds.
	C ¹ 256	Diminished.
Perception	C ² 512	
Over—35 seconds	{ C ³ 1024	
	{ C ⁴ 2048	

The testing showed that the patient was obviously suffering from a pure nerve deafness (bilateral).

As his occupation predisposed him to nerve deafness a difficulty arose, but as both ears were equally exposed they should have been more or less equally affected whereas in this case on the right side there was almost

complete deafness, whilst on the left side the hearing was not affected to anything like the same extent.

As the patient was obviously suffering from an intracranial tumor, and as the condition of the right ear could be produced by a tumor arising either from the auditory nerve or from the anterior part of the right cerebellar region and pressing forward on the nerve, it seemed obvious that this was the correct interpretation.

Mr. William Turner, under whose care the patient was admitted, came to the same conclusion.

At the operation, which was performed in two stages, a large infiltrating tumor was found, which involved the anterior part of the right cerebellar region and was attached above to the tentorium.

At the post-mortem examination it was found that the tumor had involved the nerve and had extended along it into the internal auditory meatus. On section the tumor proved to be a sarcoma. Microscopical examination of the nerve showed signs of degeneration; no evidence was forthcoming to show whether the nerve was primarily or secondarily affected.

As deafness is usually the first symptom in these cases, it seems to me that when a case presents itself in which there is severe nerve deafness, which affects one ear more particularly and for which no cause can be found, then this case ought to be very carefully examined by a neurologist with a view to excluding a tumor in this region.

It is interesting to note that the facial nerve escaped in this case and that in this class of cases it is often only very slightly involved, a condition which from its position one would have expected just the reverse.

With regard to Weber's test, I think this is just one of the cases in which it is of great value, confirming as it does the diagnosis of pure nerve deafness as opposed to oto-sclerosis complicated by secondary involvement of the internal ear.

NEGATIVE PRESSURE AS A THERAPEUTIC AGENT IN DISEASES OF NASAL ACCESSORY SINUSES, THROAT, EAR, AND MASTOID.¹

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MY apologium for this paper rests upon the fact that most continental observers who have investigated this field of work have reported results unqualifiedly unfavorable to the induced hyperæmia methods of therapy in aural, nasal, and accessory nasal affections, whereas my own experience leads me to regard it as a method of first rank. The therapeutic procedures of Wright and Bier are complementary, and the advantage of combining vaccines and hyperæmia in combating infections has already been called to the attention of the medical profession in the field of general surgery by Dr. J. C. Hollister in a very able article written about eighteen months ago. The advantages in general of such a non-operative cure of infections are so obvious as to need no mention. The conservation of affected and contiguous tissues theoretically possible along these lines would nowhere in the body show more conspicuous differences in functional end-results, as compared with those end-results previously obtained along surgical lines than in the region of aural and nasal surgery. In the middle ear and mastoid, the amount of scar tissue left by an infection cured by such non-operative procedure must

¹ Read before Academy of Ophthalmology and Oto-laryngology at Cleveland, Ohio, Aug. 29, 1908.

be infinitesimal compared with that remaining after operative cure; the loss of mastoid bony substance would be in most cases so small as to be unappreciable, and it is upon these two changes that the impairment of hearing after otitis media suppurativa and mastoiditis depends. In labyrinthine suppuration, operative interference is uninviting, to say the least. Should such a curative measure prove practicable for application to this class of aural affections, it would prove a boon of inestimable proportions. In affections of the accessory sinuses of the nose, whether of acute catarrhal, acute purulent, or chronic purulent character, the measures advocated in this paper offer possibilities of conserving essential structures of turbinates and sinuses to an extent as yet unreached by any procedures hitherto undertaken. Bier's method of applying induced hyperæmia therapeutically has been the subject of a number of reports by continental observers. Bier's views on the subject of hyperæmic therapy in otology and rhino-laryngology, as expressed by Meyer and Schmieden, are as follows: "Unassailable proof has been rendered, principally at Bier's own clinic, under the supervision and co-operation of a well-known ear specialist, that a large percentage of cases of acute otitis media purulenta with mastoiditis, as well as of acute exacerbation of old middle-ear inflammations, can be cured without any major operation. Very recent cases heal under obstructive hyperæmia alone. If inspection of the drum membrane suggests an accumulation of pus in the middle ear, paracentesis is promptly done, or a former perforation is dilated. With continued artificial hyperæmia, the fever usually drops quickly, and the inflammatory process, with its pathognomonic pain, tenderness, redness, and swelling over the mastoid, gradually subsides." "The elastic band around the neck is worn regularly for from ten to eleven hours twice per day, or uninterruptedly for twenty-two hours with two hours' intermission. A correct technique is all-

essential." "In chronic cases of otitis media or those of cholesteatoma, artificial hyperæmia without operation should be discouraged." "The same beneficial effect may be observed in the complicating acute affections of the frontal sinus and the antrum Highmori." "Obstructive hyperæmia by means of the suction glass applied through the nostrils has been extensively tried for diagnostic purposes as well as for the treatment of suppurative affections of the accessory sinuses, including ozæna. However, specialists are still greatly at variance as regards its value in these affections."

In *Monatsschrift für Ohrenheilk.*, 1906, No. 5, Fleischmann reports: 24 cases of otitis media suppurativa, 8 of which were uncomplicated, 12 with mastoiditis, 2 chronic cases with acute mastoiditis and 2 with perichondritis. He concludes that the method is dangerous on account of the fact that pain is apt to be relieved at the same time that the disease is spreading, thereby lulling patient and surgeon into a sense of false security and allowing intracranial complications to develop. He is inclined to regard the method of Bier with disfavor.

In *Archiv für Ohrenheilkunde*, lxi., Isemer reports: 2 cases of acute otitis media suppurativa uncomplicated, 1 case of chronic otitis media suppurativa uncomplicated, 9 cases of acute otitis media suppurativa with mastoiditis. His conclusions are the same as Fleischmann's.

Horslauer in the *Münchener Medizin. Wochenschrift*, 1906, No. 34, reported: One favorable result in fourteen cases of chronic otitis media suppurativa, 30% of which came to mastoid operation. He reported on the Bier method as being a distinct advance in the treatment of otorrhœa, in spite of the poor showing of his statistics, certainly not a very convincing support of favorable views concerning the method. A year and a half ago, in a paper read before the Iowa State Medical Society, the writer broached his views on the subject of applying the methods here advocated to

aural and nasal infections. The limitations of a small private practice render impossible the assembling of statistical evidence for or against the methods advocated in anything like convincing array. He can only outline the working theory of the therapeutic application of negative pressure, report his own successes and failures, and give his conception of the reasons underlying failures on the part of those using constrictive hyperæmia.

The theory upon which these therapeutic measures are based is as follows: In the presence of an infection, the body cells throw out barriers of organized inflammatory exudate round about the infected focus in an attempt to limit the spread of infection. While this barrier is of great advantage in confining infection from spreading, thus protecting contiguous structures, it has this disadvantage, that it provides a wall within which bacterial propagation and toxin elaboration can go on more or less undisturbed. Serum, the natural menstruum of the bactericidal and antitoxic elements of the body, is found within the above mentioned barrier, but the difficulties of ingress and egress cause a serum stagnation within the infected focus, as a result of which the serum of the focus suffers a loss of bactericidal potency by reason of long-continued contact with bacterial protoplasm within the focus using up these elements of its composition. This serum of lowered bactericidal potential eventually steps out and its place is taken by fresh serum of higher bactericidal potential from the general circulation; but in many instances this serum exchange takes place so slowly that the bactericidal action of the serum elements is but very slight. In other instances, there may be a more rapid serum circulation through the infected tissues and still little or no antibacterial action on the part of the serum by reason of a low bactericidal potential of the serum of general circulation. In either case, until the occurrence of some change in conditions, the tide of victory would lie with

the invaders. Attempts to bring about such changes as might turn the tide in favor of the body cells, involve consideration of the invading organism and consideration of the individual attacked. The attacks of certain bacteria are, of their own nature, of short duration. We are wont to speak of diphtheria as a self-limiting disease. Why? Because whether interfered with or not, the attack will have terminated of its own accord within certain comparatively short limits of time; at the end of which, if death has not supervened, the patient will have recovered. The attacks of other bacteria are of the opposite type. Unless successfully interfered with, the process may go on for an indeterminate period of time. The mention of bacteria under such a classification is relevant only in this, that it enables us to put out of our consideration at the start all those of the first or self-limiting type. Inasmuch as bactericidal measures suitable for application to bacteria within the body tissues have not as yet been developed to a point where decisive effects can be accomplished within the ordinary lifetime of such short-term insults, the principal fight in such instances must be directed, not against the bacteria themselves, but against their life products. This is essentially the field for antitoxins, not for vaccines or induced hyperæmia.

All bacterial infections not falling in this short-term group are fit subjects for attack along the lines of vaccines and induced hyperæmia therapy. Some along the borderline between these two classes may properly be attacked both antibacterially and antitoxically. The general condition and the specific resistance of the individual attacked by the infection have very important bearings upon the subject of antibacterial therapy. In one patient, it may be that the specific resistance to an infection will be found very low (witness low specific resistance to staphylococcus in acne subjects); in another with the same infection, it may be found very high. Vaccination is indicated in the first case, not in the second case.

Thus when using induced hyperæmia in combating infections, it will be of considerable importance in some cases to combine with this measure a judicious use of exogenous vaccine, in order to secure a high antibacterial potency of the general circulation; and in other cases such vaccination will be quite unnecessary, the secondary effect of the induction of hyperæmia proving adequate in maintaining high opsonic content in the general circulation.

There are obvious and important differences between the constrictive method of inducing hyperæmia, used by the Continental observers, and the method advocated in this paper. Constriction results in venous stasis, which in turn causes back pressure in the arterio-venous capillary anastomoses and thence in the arteries. Stasis is followed by dropsy into the tissues drained by the constricted veins. Consider for a minute what this means in chronic sinusitis of one of the accessory nasal sinuses for example. Obstructed drainage due to inflammatory changes in the sinus mucosa has already resulted in retention of muco-purulent matter with retention and pressure symptoms; possibly it has even reached the stage where the œdematous mucosa, lacking room within the cavity for its increase in bulk, has forced its way through the normal openings into the nose in the shape of polypi, still further obstructing the exit of pus from the cavity. Increase by constriction this already obstructive dropsy, and it is small wonder that effects of an unfavorable rather than of a favorable nature should be noted. The same unfavorable effect of constriction should be expected in acute mastoiditis, in which condition alarming symptoms are always due to blocking up of the exits of the infected cavities, rendering drainage insufficient. Freeing the exits of all such cavities, not only of polypi, but of all swelling of adjacent tissues and mucosa, removing all secretion possible by a continuous stream of warm alkaline solution, which tends to dissolve it and

syphon it from within the cavity, using from two to four quarts of lavage solution at a sitting, we open up our exits as widely as possible. Following this up by negative pressure, which sucks out of the cavity a large amount of the retained secretion and sucks into the tissues lining the cavity a sudden great excess of fresh serum and lymph and phagocytes, we have not only still further increased our drainage by mechanical means, but we have dislodged stagnant lymph and serum of low opsonic value and brought in an excess of lymph and serum of high opsonic value. After these changes in the tissues have been accomplished, the treatment is at an end. The elasticity of the tissues allows a reactionary subsidence of the acute distension caused by the negative pressure. In the very nature of things such subsidence can occur only by the moving forward into the general circulation of the excess of fluid which has caused the distension, as there is possible no backward outflow. So it must result in the outflow of the *old stagnant fluids* whose opsonic potential is already exhausted, and the replacement of those fluids by lymph and serum of high opsonic potential.

CASE I.—March 12, 1907, male, eighteen years, brought by family physician for operation. Acute otitis media suppurativa of one week's duration. Bacteriology: *B. lanceolatus* and pus cocci. *Status præsens*: Pain, prostration, fainted twice to-day; slight sanguinous pus discharge through punctate perforation in ant. inf. quadrant of membrana tympani. Mastoid swelling and extreme tenderness. Temperature 103°. Anæsthetized; free incision from floor up through memb. flaccida into attic. Suction applied for ten minutes, after which patient was kept at absolute rest, with occasional application of hot-water bag to mastoid. Suction was repeated daily. The following 72 hours, temp. fluctuated between 102° and 99°. Pain was practically nil; discharge free. Two days later, temperature became normal. Swelling and tenderness in mastoid disappeared. Patient gained strength and felt almost well. Discharge had

ceased by the thirteenth day, when patient went to work. A month later hearing was full normal.

CASE 2.—March 24, 1907, male, thirteen years. Mastoiditis complicating acute exacerbation of chronic otitis media suppurativa A. S. following scarlet fever at four years of age. Has had occasional exacerbations of mild character, never before as severe as this one. Profuse discharge of thick, intensely foul pus for three days, followed by diminution in amount of discharge, great increase of pain, œdema and extreme tenderness over mastoid. Auricle stands out at angle of about 30° . Teat-like pouting perforation in ant. inf. quadrant of *Mt.* Temp. 102.8° ; prostration and marked febrile symptoms. Bacteriology: Staphylococcus and pneumobacilli. Incision carried from floor through perforation deep into attic. Suction applied for ten minutes. During first forty-eight hours temperature fluctuated between 103.8° and 100° and pain disappeared under daily suction; after fourth day, temperature remained normal and patient could come to office daily where suction was applied for from eight to twelve minutes. On the eighth day all mastoid œdema had disappeared, some tenderness still remaining. On the fourteenth day, began instilling absolute alcohol after applying suction. This was continued daily for two weeks, when discharge had almost disappeared. Very large faucial tonsils were enucleated and naso-pharynx curetted. Suction applied twice weekly until, two months and a half after coming into my hands, all discharge had ceased, lower anterior quadrant of drumhead healing partly adherent to inner tympanic wall. Hearing improved from inability to hear thirty-six-inch watch on strong pressure, to watch at eight inches. One year later heard same watch at twelve inches.

CASE 3.—May 10, 1907, female, twenty-two years. Acute otitis med. sup., two days no perforation. Temp. 99.6° , intense pain, no mastoid symptoms, made free incision of drumhead. Bacteriology: *B. lanceolatus*. First twenty-four hours patient was much improved, during following ten days improvement was interrupted by occasional pain in ear and some mastoid tenderness. By twentieth day discharge had almost ceased. At this time symptoms of an exacerbation presented, perforation began to pout, mucoid

secretion changed to finely granular pus, and mastoid pain began. Made free incision from floor to attic and applied suction for ten minutes, evacuating an enormous amount of pus; temperature has been normal since tenth day. Eight days later I was compelled to make an extensive simple mastoid operation, finding granulation tissue and pus from tip to zygomatic cells. Healing was uneventful.

CASE 4.—December 9, 1907, female, twenty-three years. Patient presented furunculosis of post. inf. canal wall; had been previously operated for acute mastoiditis, simple operation having been done. During this operation I found it necessary to curette through a small portion of post. bony canal wall, the site of which dehiscence chanced to be that selected by the present furuncles. After thirty-six hours of intense pain, during which time post. wall was so swollen as to hide all view of the tympanic membrane, patient experienced sudden spontaneous subsidence of all symptoms, including swelling. The next twenty-four hours, explanation of this unlooked-for eventuation of the ext. otitis became apparent. The old mastoid scar became reddened, œdematous, and tender; temp. 102° ; when suppuration had occurred beneath the cuticle-periosteum, pus found ready escape backward through the dehiscence of the post. bony canal wall into the old healed mastoid cavity. I at once incised the old scar, incision being followed by free discharge of pus. Bacteriology: *Streptococcus* and *pneumobacilli*. In spite of drainage mastoid continued to discharge through scar incision for over a month, patient feeling well and strong however, and pursuing occupation as high-school teacher. I made many applications of negative pressure to discharging sinus during the next four weeks, but failed to secure cessation of discharge, though there was great diminution in amount of pus. I finally operated, cleaning out old mastoid cavity, incising *Mt* which had been intact, and establishing gauze drainage from cavum epitympanicum backward to mastoid incision. Healing was interrupted by a week of facial erysipelas, after which it was uneventful. Hearing a year after first operation had been eight inches for thirty-six-inch watch. Now, six months after this second operation, she hears same watch only $\frac{1}{2}$ to $1\frac{1}{2}$ inches. My first two cases of acute mastoiditis had

responded so nicely to treatment by negative pressure, that I was quite a little disappointed at being compelled to operate on the subjects of my next two applications of this method. Case 4, however, I was inclined to consider hardly a fair test on account of the entire change in the local circulatory and cellular conditions in this region, due to the previous operation. I decided, however, after these two experiences, to make a longer application of negative pressure to the next acute mastoiditis so treated. And in this connection let me make it clear that I have not applied this treatment to every acute case, nor do I consider it adapted to all acute cases. There are certain cases which early present the picture of quickly spreading virulent infection with rapidly deepening intoxication, intense and steadily increasing prostration. These cases I would certainly operate at the earliest possible moment without attempting any conservative delay.

CASE 5. March 19, 1908, female, twenty-eight years. Had been treated for acute catarrhal Eustachian salpingitis three months ago, which gradually improved without perforation of *Mt*. Five days ago developed acute otitis med-sup. in same ear. Temp. 101.4, thick purulent discharge for past three days, mastoid swelling, redness and tenderness. Anæsthetized and incised *Mt* from floor through memb. flaccida. Applied negative pressure for twenty minutes, evacuating enormous quantity of sanguinous pus. At end of twenty-four hours temp. 101.8, headache, malaise, severe pain. Anæsthetized again and reopened incision, applying negative pressure forty minutes. Bacteriology, diplobac. lanceolat. In twenty-four hours temp. 99.2; felt much improved; free discharge from drumhead incision. Two days later applied negative pressure five minutes without anæsthetic and repeated this cautiously every day for next week. Except for an intercurrent furunculosis of canal, patient made an uninterrupted recovery. Six weeks after first operation, hearing for thirty-six-inch watch was twenty-four inches.

CASE 6.—Female, sixty-two years. Chronic sup. otitis med.; extremely foul pus, discharge dating back to scarlet fever in childhood. View of drumhead obscured by large polypi; these being avulsed large perforation in inf. post.

quadrant was exposed. Applied negative pressure without anæsthesia for ten minutes, followed by 95% alcohol instillation. Repeated neg. pres. and alcohol daily. A week later avulsed more polypoid tissue. At the end of twenty-four days perforation had entirely closed, having contracted adhesions with inner tymp. wall. Hearing had improved from not hearing thirty-six-inch watch on firm pressure, to hearing same watch at three inches. Perforation has remained absolutely closed for four months.

CASE 7.—April 17, 1908, male, thirty-nine years. Exacerbation of chronic catarrhal sinusitis, three days, referred to me by family physician. Temp. 100.8, has reached 103. Intense pain in head, particularly on left side, in inf. max. and frontal regions, tenderness on pressure over frontal and inf. max. sinuses. Has used warm Seiler's douche for past three days with slight relief. Pain has grown steadily worse; gr. $\frac{1}{4}$ morph. hypo. necessary this A.M. Applied cocaine to swollen turbinates and contiguous mucous membranes and applied negative pressure, evacuating thick pus. Bacteriology, pyocyaneus and pus cocci. Relief of symptoms was immediate, within three minutes. After full shrinkage of mid. turbinates found large true hypertrophy of ant. end right mid. turb. which I snared off at once. The left had been the larger of the two and most of pain had been on left side, but after cocainization there was no bogginess, so this was not touched. Ordered continuation of Seiler's douche. Relief of pain was complete from 10 A.M. until 3 A.M. next morning, when it again became so intense as to require morphia. Neg. pressure applied at 9 A.M. after cocainization and relief of yesterday again experienced. This was continued daily for about a week, then successively thrice weekly, twice weekly, once weekly until by the end of six weeks no pus had been seen for a week or more, nothing but non-purulent mucus, and this had about ceased to appear in appreciable quantities. Since the third day patient had been free from pain and has been able to go back to his work as supervising architect.

CASE 8. Male, thirty-five years. Had been under my care off and on for nearly three years on account of chronic catarrhal sinusitis involving frontal ethmoidal and sup. maxillary

sinuses on each side, occasionally worse on right, occasionally on left. Patient had repeatedly refused proposed operative measures for relief of intranasal conditions, which included very thick deflected septum. Had also been in the hands of other rhinologists occasionally, all competent men, and had once submitted to some turbinate operation at the hands of one of these men, upon which ensued an unfortunate hæmorrhage which only served to confirm him in his opposition to subsequent operative proposals. He had run the gamut of frontal and maxillary lavage, topical applications to infundibula and turbinates, alkaline nasal lavage at my hands, and of catarrh inhalers, jellies, etc., at his own hands, without signal relief. On the occasion of one of his excruciating headaches during a purulent exacerbation of his trouble, I packed the infundibula with cotton saturated with cocaine solution as I had often done for him, and after thoroughly shrinking the tissues, I applied negative pressure to the nares. The effect was particularly satisfactory because of the fact that it was comparable with effects of many other procedures applied under similar conditions on the same patient; within five minutes after evacuating a large quantity of greenish pus, all pain had disappeared. Pursuing this plan of treatment, at first daily, later thrice weekly, twice weekly, and at last once weekly, I was able to keep the patient almost free of headaches and that sensation termed "wooden-headedness," "balloon feeling," etc. Three subsequent purulent exacerbations have since been successfully aborted by early application of negative pressure.

CASE 9.—Female, sixteen years. Was referred to me by family physician because of headaches. She had had gastrointestinal disturbances and he had been inclined to attribute the headaches to this source, though treatment for them along this line had proven unsuccessful. She was highly myopic, wearing 4.50 sph., and was of a catarrhal type. I found she had a history of frequent attacks of tonsillitis and pharyngitis and had recently had a mild otitis med. suppurativa. Curettement of the nasopharynx and enucleation of faucial tonsils was first done, after which I turned my attention to the nose, which was in a condition of marked hypertrophic catarrh. After shrinking the structures contiguous to the infundibula

and washing thoroughly with a large quantity of warm alkaline solution, I applied negative pressure for about eight minutes and succeeded in evacuating a large amount of very thick stringy yellow pus, most of which came from the sup. max. sinus, to the great surprise of the patient, who declared she had never been aware of the presence of such secretion. Her discomfort and headache promptly ceased after the discharge and she remained free from symptoms for two days, when I reapplied the negative pressure with same results. This course of treatment was continued at first thrice weekly, later twice weekly, at last once a week. The character of the evacuated secretion changed to mucus and the amount gradually diminished to a normal quantity. Occasionally after exposure she suffered slight exacerbation of symptoms, with a tendency toward increase in quantity and purulence of secretion, but her general progress was steadily towards what seems now to be a complete cure.

CASE 10.—Female, twenty-two years. April 22, 1908. History of morning headaches for over two years, often so severe as to prostrate her. Pain comes on about 8 or 9 A.M. and lasts till from 1 to 4 P.M.; vomiting occurs frequently at the height of the pain but slight relief of pain is experienced afterward. Narrow high arched palate, thick septum, very much hypertrophied nasal mucosa showing true general papillary hypertrophy. Lower and mid. turbinates in contact with septum. After shrinking the infundibular regions as much as possible, negative pressure evacuated much tenacious yellow pus containing pus cocci and some diplobacilli. Patient experienced great relief at once. I pursued the usual course of thrice weekly and later biweekly applications of this treatment. After the first four weeks had passed in comparative comfort, patient having had only an occasional abortive attack of headache, while superintending moving of her household goods to a new home she was exposed and suffered sudden exacerbation, chill, temp. 102, intense pain in head, nausea. She came at once for a treatment at 2.30 P.M. I was called at 7.30 P.M.; found her frenzied with pain, temp. 102.8. I was compelled to give morphia gr. $\frac{1}{2}$ hypo. in two injections two hours apart before there was any appreciable diminution of symptoms. Later, after being confined to bed

for three days by this attack, patient resumed treatments every day with very unsatisfactory results. The relief she had experienced at the beginning of the treatments was conspicuously absent and at times I suspected her discomfort had been actually increased by the negative pressure. After a week of non-success, I advised her to give up all treatment and watch developments with a view to some operative procedure in case symptoms should demand interference. Five weeks later she reported having been almost entirely free from symptoms and she had had no bad headaches. After four months patient reports having had no return of headaches though she has occasional discharge of large amount of thick mucoid secretion. In this case I am somewhat at a loss to account for the apparent increase in symptoms while under treatment, promptly followed by disappearance of all discomfort on discontinuing negative pressure. It may be that absorption of the inflammatory hyperplastic tissue in the sinus mucosa was interfered with by the frequently induced congestions and that on ceasing to induce hyperæmia such absorption did occur with beneficial subjective results. I am more inclined to believe that this case was one not well adapted to treatment along these lines and that the improvement noted just now is only a temporary fluctuation toward betterment, such as is shown by most cases of chronic sinusitis suppurativa.

CASE II. Male, forty-six years. Has had chronic catarrhal rhino-sinusitis for years, has been cauterized, chromicized, etc., at different times. For past few weeks has had balloon feeling in head, morning headache, discomfort in frontal ethmoidal and sup. max. regions, slight tenderness on pressure, with more or less obstruction to nasal respiration. Somewhat irregular septum, boggy turbinates with little true hypertrophy. Infundibular structures were well shrunken, after which warm douche and negative pressure were applied. Evacuation of considerable grayish thickened mucoid secretion was followed immediately by relief of symptoms. Treatment was continued with gradually increased intervals until within three weeks there was no recurrence of symptoms and patient felt perfectly well. Patient, who is a physician, is extremely enthusiastic over the success of this method of treatment,

declaring it to be far the pleasantest and most efficacious treatment he has ever experienced.

CASE 12. Female, twenty-six years. Has well marked beginning atrophic rhinitis with usual purulent sinusitis; crusting and odor have been source of great discomfort to patient. Has been treated for over a year by topical applications and lavage without improvement. Warm alkaline lavage followed by a negative pressure applied daily for ten days. At first free hæmorrhage occurred at each treatment and a large amount of foul yellow thin muco pus was evacuated. Hæmorrhage ceased after five or six treatments, discharge grew less in amount, lost its foulness, becoming more and more mucoid. Subjective symptoms ameliorated, odor disappeared. After eight weeks, during which intervals between treatments were gradually prolonged, patient was so far improved that she was instructed to stop treatment, using only warm lavage at home once or twice daily, and to return in case there was any increase in symptoms. I have not seen her in over two months, but she reports no return of odor or of sufficient crusting for her to be conscious of its presence.

In summing up these cases together with about twenty other cases not detailed, because of a desire to avoid unnecessary and tiresome repetition, I must report that the results of applying induced hyperæmia therapy in the manner described have been so satisfactory in my hands that I cannot but regard it as a therapeutic method of indubitable worth. It is not recommended as applicable in all cases. In properly selected cases, it is of superior merit in that it offers a cure without the operative hazard, in many cases without even the hazard of a general anæsthetic; and it is of especially superior merit in that it offers a cure with the least possible loss of tissue and disturbance of anatomic relations and consequently the greatest possible conservation of function.

Applications of negative pressure for relief of purulent sinusitis should always be preceded by thorough shrinkage of intranasal structures. The technique of applying

negative pressure to the nose and ear is simple and needs no detailed description. Any practical means of maintaining continuous suction may be made use of. An ordinary air-pump operated by water or electric power seems best adapted to the purpose. In the office I use the chemist's vacuum water-pump suggested by Dr. Frank Brawley, of Chicago, to whom I wish to acknowledge a deep indebtedness for having brought to my attention this excellent device for applying negative pressure in these regions. For portable use I have impressed into service an old glass ear syringe of 100cc capacity. In using either pump or syringe, it is better to use non-collapsible tubing rather than the ordinary rubber tubing. To the intake tube is attached a suitable tip of glass or hard rubber. The Siegel otoscope is very satisfactory for use in the ear. The olivary tip or the glass tip devised by Dr. Will Walter are best adapted for use in the nose. The nostrils are occluded, one by inserting the nasal tip, the other by pressure from the patient's thumb. The negative pressure is then applied and at the same time the patient is instructed to swallow, to start the act of saying K, or to open the mouth very widely. Any one of these acts will bring the soft palate up against the posterior pharyngeal wall, and the negative pressure will hold it in this position, thus closing off the posterior openings of the nasal chambers and allowing the partial vacuum formation in the nares. A great advantage of the water vacuum pump over other methods of inducing negative pressure is the absolute control allowed the operator over the amount of negative pressure applied. By opening or closing the water-cock, the negative pressure can be varied immediately according to the desire of the operator. After a considerable experience in using this treatment in purulent sinusitis, I have come to realize that posture plays a very important part. I have often been unable to evacuate pus from a maxillary sinus, for example, while the patient is in an upright

position, only to find that by placing the head for several minutes previous to the application of the negative pressure in such a position as to cause the contents of the sinus, by gravitation, to overlie the natural opening or openings, an enormous discharge of thick pus follows immediately upon the reapplication of even slight negative pressure in the new position. I would lay special stress upon the importance of posture and of non-collapsible tubing. It is convenient to have a small bit of glass tubing inserted a few inches away from the tip so that the operator may observe the passage of pus from the nose. It is also a great convenience to have a four-ounce wash-bottle at some convenient point along the tube for collecting the secretion removed.

PHLEBITIS WITHOUT THROMBOSIS AS A CAUSE OF OBLITERATION OF THE SINUS IN CHILDREN.

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Abridged translation by Dr. GERHARD H. COCKS, New York, from
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A NUMBER of cases of obliteration of the sigmoid sinus have been described in literature. Streit mentions this condition as an accidental finding at operation. Hölscher twice found granulation tumors instead of sinus, and once met with a sinus degenerated into granulation tissue; he thought that there had been a previous sinus phlebitis with destruction of the vessel. Still other cases of sinus obliteration have been reported in the Austrian Otological Society (Dec. 30, 1905) under the heading "Healed Sinus-Thrombosis" (Alt, Alexander, Fry). Further observations have been made by Warnecke, Zaufal, and others.

We thus see that it is not very rare for an inflamed sinus which has not received operative treatment to lead to obliteration of the sigmoid. That this must always be caused by a previous thrombosis of the sinus, is a supposition which is evidently quite generally held. Hoffman's case may be quoted as a classical example.

In a four-year-old child, with abscess and cholesteatoma in the mastoid, ichorous fluid escaped from the posterior cerebral fossa through a defect in the posterior wall as large as a dime; there was also an extra-dural abscess present.

Division of the sinus showed the wall much thickened; the lumen existed for a distance of 1 cm only and contained blood-tinged discolored fluid. Centrally and peripherally from this point the sinus was obliterated by firm connective-tissue adhesions between the walls.

The same conditions were present in Case 1 reported by Warnecke, who observed obliteration of a sinus in both directions by a completely organized connective-tissue thrombus. There was an abscess lying on the sinus.

However, in some other cases of obliteration of the sinus, an inflammatory thrombosis cannot be brought forward as the cause. Here Case 2 of Warnecke belongs

He opened the mastoid of a three-year-old girl, and found a moderately large cavity filled with foetid pus and granulations, in the bottom of which the sinus was exposed, covered with granulations; further down the sinus was covered by a sequester. When opened, it was seen to be free from blood from the mastoid tip to the knee. After passing the sound posteriorly moderate bleeding occurred; no bleeding anteriorly, where it was impossible to follow up the vessel further because it was completely obliterated in the region of the tip. No organized connective-tissue masses of thrombotic material could be recognized between the sinus walls.

In Warnecke's opinion either previous phlebitis caused primarily the agglutination of the walls after the lower portion of the sinus had collapsed in consequence of a thrombus located above, or the agglutination was secondary to disintegration of a thrombus which had existed at this point. Warnecke assumes that a thrombus was the direct or indirect cause of the gluing together of the walls.

Muck found a sinus collapsed in the sigmoid groove, in a child nine years old with a large extradural abscess, extending over a portion of the occipital lobe and cerebellum. Healing resulted quickly without the sinus being opened. Muck thought that the sinus was without

doubt thrombosed in the place where it was covered with granulations in the region of the abscess, since it was in a collapsed condition further anteriorly, *i. e.*, centrally.

Leutert's case—a seven-year-old girl who had been ill with scarlet fever $3\frac{1}{2}$ months before—was successfully operated on account of necrosis of the mastoid process and a purulent fistula 6cm behind the insertion of the auricle leading into the mastoid emissary. The sinus could not be recognized. It was not possible to establish the occurrence of a previous pyemia from the history.

These three cases have the following points in common: They occur in children whose ages are seven, nine, and eleven years respectively; an extradural abscess was either present or had existed presumably before the destruction of the bony sulcus; and all three healed without interference with the sinus itself.

Obliteration of the sinus may take place in still another way than through an inflammatory thrombosis, viz., through compression and phlebitis by an extradural abscess with resulting agglutination of the vessel walls. The two following cases observed by me are examples:

CASE 1.—H. F., six years old, 1903; scarlet fever, followed by left-sided otorrhœa till May, 1906. On July 2, 1906, profuse discharge left ear, severe pain over left half of head, vertigo, high fever, no chills. Admitted to hospital, July 13, 1906.

Examination.—T. 38.7° P.M. *R. ear:* Moderate purulent secretion in canal; heart-shaped defect in lower part of drum; upper half thickened and somewhat reddened; tympanic mucosa red. *L. ear:* Auricle at right angles to scalp; soft parts over mastoid swollen and tender upon pressure; fluctuation over mastoid fossa; in external canal abundant purulent secretion; canal narrowed by collapse of superior wall so that only the posterior part of the drum was visible, here bulging and red. Background of eyes normal. Moderate-sized adenoid. Paracentesis, left. Removal of adenoid.

July 14, 1906.—Mastoid operation, left. Periosteum thickened; 3cc pus between bone and periosteum; bone intact;

numerous large bloody spots in mastoid fossa; mastoid cells filled with bloody swollen mucous membrane. In the antrum was a slight amount of pus and swollen mucosa.

During the two following weeks the patient had slight fever, but nevertheless gained in weight. She remained in bed on account of the persistence of moderate discharge from the ear. On August 14, when she left her bed for the first time, the T. shot up to 39° , and sharp pain was experienced behind the left ear. On August 19, T. was 37.1° A.M. and 38.1° P.M. Eyes normal; general condition good. Moderate purulent secretion in canal. Slight tenderness behind the field of operation.

August 20.—Secondary operation. Wound scraped. The corticalis behind the mastoid was brittle and roughened over an area the size of a shilling. When removed with the internal table 5cc of pus was evacuated. The dura of the posterior fossa was covered with granulations, necessitating wide removal of the bone. After the granulations were removed the dura was apparently healthy except in one small sharply defined spot, where it was yellowish-green in color. Next to this, corresponding to the course of the sinus, the granulations were firmly attached to the dura. When the apparently destroyed dura was removed, I came upon a sinus completely empty (just below the knee). Anteriorly and posteriorly from this point the lumen was obliterated, the walls being adherent. There was no thrombus present.

The further course was uneventful, apart from a light attack of erysipelas. After a few days the secretion from the ear disappeared. When examined about two months after operation, the retro-auricular wound and the perforation in the drum were closed. Hearing for whisper fairly good.

CASE 2.—W. R., five and a half years old; no previous ear disease. July, 1906, measles. Early in August, discharge and then pain, first in the right and then in the left ear. Since August 21, 1906, increasing swelling behind left ear without appreciable impairment of general health. Admitted to clinic August 24.

Examination.—*Right ear:* Moderate tenderness over mastoid fossa; slight amount of pulsating purulent secretion in canal; posterior part of drum membrane red and bulging;

Paracentesis. *Left ear:* Tenderness upon pressure over mastoid fossa; small fluctuating swelling just behind this point, limited posteriorly by the posterior mastoid border, and accompanied by infiltration of the soft parts; moderate purulent discharge in canal; drum completely absent, except for a triangular piece above which contains remnant of malleus; tympanic mucosa thickened and red. Background of eyes: Papilla somewhat reddened and sharply defined. General condition good.

Operation.—Left mastoid opened; subperiosteal abscess. Behind mastoid fossa is a bony fistula 0.5cm broad. After enlarging the fistula and scraping away the granulations, I came upon the white tabula interna of the sulcus which lay completely free over an area 2cm long and 1cm wide. The antrum and mastoid cells were filled with greatly swollen and hyperæmic mucous membrane, but contained no pus. While examining the internal table of the sulcus, a drop of pus appeared. The internal table yielded slightly to my pressure, and finally this portion came away in toto as a sequester. This was followed by 3cm of pus, which emptied itself from the interior of the cranial cavity. The sinus wall and surrounding dura were covered with flabby granulations. In the middle of the granulations was a linseed-sized perforation in the outer sinus wall; the inner wall of the sinus, grayish-white and thick, lies free. The lumen of the segment of the sinus adjoining the opening was obliterated by agglutination of the walls. Recovery uneventful.

In both these cases we have to do with an acute mastoiditis, which originated without any special cause in Case 1, while in Case 2 it followed measles. In Case 1 the middle-ear suppuration on the diseased side was said to have existed for three years, yet the examination and clinical course make it probable that the opposite ear was affected with the chronic suppuration, while the operated ear was probably healthy until a short time before. In Case 2 we are dealing with an acute exacerbation of a chronic middle-ear suppuration.

Both patients developed an extradural abscess and

sinus phlebitis in connection with an acute mastoiditis. In each case the cause was an inflammatory focus in the bone. In the first case the diseased area remained after the mastoid operation. In Case 2 there was no previous operation on the antrum. The inflammation within the antrum and contiguous cells receded by draining through the large defect in the drum. In the distant cells, corresponding to the upper posterior portion of the mastoid, the infectious material had opportunity to do its injurious work.

The symptoms which point to extradural abscess and sinus phlebitis, apart from the unusual site of the diseased bone, were the following:

In Case 1, after the mastoid operation a moderate rise of temperature to 38.0° C. and over was observed during the evening for a month. When the child left its bed for the first time the fever reached 39.4° C., severe pain was experienced behind the operative wound, and a profuse discharge from the middle ear made its appearance, after the canal had been dry for several days.

Case 2 was operated the day of admission to the hospital. Here there were no symptoms worthy of note. Certainly the general condition and the temperature did not point to any severe complication.

The essential point in both cases is that it was possible to observe the manner of healing of an inflamed sinus in the natural way.

Through disease of the bone in the neighborhood of the sigmoid sulcus, the wall of the sinus was either inflamed or perforated by the pus. Thus there originated a perisinus abscess and an inflammation of the outer sinus wall, with the formation of granulations. The abscess and granulations, in spite of the slight amount of pus, led to flattening of the sinus, thus making it possible for only a slight amount of blood to circulate. The inflammation of the outer wall gradually progressed from the superficial to the deeper layers, until finally all layers

of the vessel wall were involved and increased in thickness. When the innermost layer became diseased, the lumen was no longer present. Thus we explain the coalescence of the inner surfaces of the sinus from the operative findings.

As the inflammatory narrowing of the vessel lumen increased and the walls began to adhere, the venous blood could no longer pass, and was shut off from the diseased portion of the sinus. A thrombosis would have resulted if the lumen had been patent up to the time that the innermost layer of the sinus took part in the inflammatory process. But the lumen was either previously or simultaneously obliterated—therefore there was no thrombosis.

What course each case would have pursued without operative interference is naturally difficult to state. Possible complications are rupture of the pus externally through the soft parts, leptomeningitis, or brain abscess. However, it could never have come to a pyæmia or sepsis in consequence of sinus phlebitis.

Through the report of my two cases, I should like to emphasize the fact that sinus phlebitis may lead to obliteration of the vessel without the occurrence of a previous inflammatory thrombosis. I believe I have demonstrated this as far as it is possible by examination upon the living.

If a thrombosis has not taken place in an inflamed and diseased sinus, a certain amount of danger exists—not necessarily very great—as long as the blood circulates through the diseased channel. The prognosis in a case of sinus-inflammation which leads to a simple permanent closure is good, in contradistinction to the outlook where there is thrombo-phlebitis, provided the diseased bone is removed and the pus drained.

My patients were children five and six years old respectively. In the cases of Warnecke, Muck, and Leutert, mentioned above, the obliteration of the sinus may well

have resulted from sinus phlebitis and compression in consequence of an extradural abscess without inflammatory thrombosis. These patients were also children, between seven and eleven years old. As far as these few instances warrant conclusions, it appears that the course of sinus disease portrayed above occurs preponderantly, if not entirely, in childhood. The explanation is to be found in the anatomic relations.

The sigmoid groove in children is considerably flatter than in adults. In the latter, the outer wall of the sinus on cross section has about the form of a half-circle; but in children, on the contrary, it is shaped more like a flat arch, while the inner wall of the sinus stretches almost straight across the sulcus. In children, then, the outer wall of the sinus lies considerably nearer the inner wall than in adults. If an abscess presses the outer wall in an inward direction, the lumen is obliterated earlier in children than in adults. Given the same pathologic changes in the region of the sinus, when the inflammatory process has involved the innermost layer of the external wall, in children there results an inflammatory sinus-obstruction by obliteration of the lumen; while in adults an inflammatory sinus thrombosis ensues, on account of the persistence of the vessel lumen.

This consideration raises the question: Does not the simple, *i. e.* non-thrombotic, sinus-obstruction occur fairly frequently in childhood?

The answer to this question must be determined in the future.

THE TREATMENT OF ACUTE MIDDLE EAR
SUPPURATION, WITH NIPPLE-SHAPED
PERFORATION, BY ASPIRATING THE PUS
INTO THE EXTERNAL MEATUS.

By DR. O. MUCK, OF ESSEN (RUHR).

Translated from *Zeitsch. f. Ohrenhkl.*, Vol. LVI., No. 1, 1908, by Dr.
M. J. BALLIN, New York.

IN the 9th number, 1907, of the *Münchener Med. Wochenschrift*, the writer reported his observations on the influence of the hyperæmic treatment, produced in the external meatus by aspiration, on suppurations of the tympanic cavity, and called attention to the fact that the suction method proved very beneficial in suppurations in which the perforation in the drum was unfavorably situated, and especially in those forms in which the perforations had a nipple- or cone-shape. Owing to the practical interest which such cases afford, the writer would like to go more fully into the details of this new method of treatment, which, as it appears, may be looked upon as the most advantageous.

If we take into consideration the pathological changes which take place in middle-ear suppurations, we may understand why the acute middle-ear suppurations associated with bulging of the drum at the posterior superior quadrant have an unfavorable prognosis. Between the long process of the incus, stapes, opening into the antrum, and medial wall of the tympanic cavity, there is a loose connective-tissue net-work, which swells up considerably

during inflammatory processes, and the meshes of which become filled with pus (similar to a wet sponge); this separates the larger portion of the tympanic cavity from the pneumatic accessory spaces which are usually also diseased, so that, in spite of perforation or paracentesis of the drum, there is pus retention. This retention often necessitates, therefore, the opening up of the mastoid process (Kümmel.)¹

A far more important and harmful factor is that the epidermis of the drum forces its way into the small perforation which in itself is very narrow, during the time that the cone is developing, whereby the opening is made still smaller, as has been shown by Katz.²

That dilatation of the narrow cone-shaped perforation or the making of a counter-opening (Schwartz) does not always bring about the desired result, is experienced by every practitioner. More satisfactory results are obtained by removing, or, in other words, crushing the projections (Haug). Aspirating the inflamed tissue of the tympanic cavity through the external meatus, by means of my suction apparatus is a method which does less harm to the parts, and which may be said to abort the process.

Amongst one hundred and fifty cases of acute, purulent middle-ear inflammation, I have observed five cases with a marked cone-shaped perforation in the posterior superior quadrant during the subacute stage. These cases recovered in about eight days under the suction treatment. The histories of these cases are briefly as follows:

I.—F. V. Thirty years old. Suppuration from the ear for three weeks following an attack of influenza. Patient

¹Kümmel, "Über die Ursachen des verschiedenen Verlaufes der akuten Mittelohreiterungen," *Verhandl. d. Ges. deutsch. Naturf. u. Ärzte*, 1904.

²Katz, "Zur Frage der bei akuten eitrigen Mittelohrentzündungen vorkommenden Trommelfellzapfen, deren Therapie und histo-pathologische Struktur." *Archiv f. Ohrenheilk.*, vol. ii, Parts 3, 4.

complains of an intense feeling of tightness in the head. A cone-shaped perforation, behind and above. After three applications of the suction method, whereupon a large quantity of pus could be aspirated through the perforation, recovery took place within five days.

II.—K. G. Eleven years old. Pain in the ear for fourteen days, marked bulging and redness of the drum, behind and above. Paracentesis. Profuse discharge of pus. After three weeks a distinct cone-shaped perforation developed. Mastoid process painful on pressure. Renewed pain in the ear. After daily aspiration of the pus, recovery and closure of the perforation took place at the end of eight days.

III.—G. N. One year old. Persistent suppuration for five weeks in spite of early paracentesis. Occasional retention of pus, accompanied by fever. A marked cone-shaped perforation, which developed during the last few weeks. Recovery after ten days, with closure of the perforation. Aspiration was performed, in this case, every other day.

IV.—F. N. Eight years old. Profuse purulent discharge from the right ear. Cone-shaped perforation. On the second day, a large quantity of pus was aspirated, whereupon the cone retracted entirely, and a perforation could not be detected even with a magnifying glass.

V.—P. W. Twenty years old. Acute suppuration of the ear of several weeks' standing. Cone-shaped perforation, behind and above. Pus, tenacious and mucous in character. After five applications of the suction apparatus, the profuse purulent discharge ceased entirely. Closure of the perforation.

The writer would like to state the following in reference to the technic. It is essential that one performs the suction intermittently, that is, one aspirates several times at one sitting. The conical portion of the glass suction tube which has been devised by the writer is hermetically introduced into the external meatus, the rubber air-bag, which is of moderate size, is then compressed, the outer opening of the suction tube is closed with the left index finger, and the air-bag is allowed to expand again. After several seconds, one lifts the index finger and upon re-

moving the glass tube one is able to see a purulent discharge in the meatus which was previously well cleansed. Every time the meatus is sponged out, pus can be aspirated 5-10 times or oftener. Aspiration is no longer continued if the exudate assumes a hemorrhagic color. This method is carried out daily. The idea that the narrow perforation becomes occluded by the suction is purely theoretical. The writer became convinced from the cases treated by him that fluid can be aspirated at any time. The writer never enlarged or removed the cone-shaped protrusion on the drum.

That such cases, which are supposed to have an unfavorable prognosis, recover in so short a time, depends firstly on the fact that the tympanic cavity is freed of a large part of the purulent fluid; secondly, that the tissue infiltrated with pus, just as a swollen sponge, to use the simile of Kümmel, is deprived of a certain amount of fluid; and lastly, that the epidermis of the drum which is turned into the perforation is drawn out by the suction. In two cases, the writer saw the cone-shaped protrusion considerably lessened on the following day, after several applications of the suction apparatus at one sitting.

Aspirating with a strong air-bag is also not painful, as one learns from the statements of adult patients. Children who cry habitually, naturally, also cry during this manipulation.

The physical action of the removal of the fluid from the tympanic cavity through the perforation, by means of aspiration in the external meatus, can be clearly appreciated from the description of the following apparatus.

A glass tube, one end of which is enlarged, is divided by a glass partition which has an opening similar to a cone-shaped perforation. The dilated portion represents the tympanic cavity; the partition, with the cone-shaped orifice, the perforated tympanic membrane; and the remaining portion the external meatus. If one fills the supposed tympanic cavity completely with a fluid which

has nearly the consistency of pus, as for instance glycerine or the yolk of an egg, so that no air bubbles are visible in the bulb, one can aspirate at highest one drop, even if repeated several times. If, however, a few air bubbles are present, then one can aspirate, if performed intermittently, an amount of fluid up to the level of the opening in the partition. If the dilated portion is stuffed with cotton, which has been impregnated with the yolk of an egg, one sees, after each suction, one or more drops of fluid exude, and after cessation of the aspiration, one or more air bubbles rise in the dilated part through the cone-shaped opening, a well-known physical phenomenon. We therefore have without doubt in the living the same physical action, inasmuch as the tympanic cavity, in suppurative inflammations, is never completely filled with fluid; this can be objectively proven when there is an exudate in the middle ear, in that the air bubbles which enter the Eustachian tube during an act of swallowing, blowing the nose, yawning, and screaming can at times be seen by otoscopic examination. This experiment, therefore, has proved that it is physically possible to aspirate fluid from the tympanic cavity through an opening in the drum. After emptying the middle ear of some of its inflammatory fluid, the absorbing action of the hyperæmia (Bier-Klapp) brought about by the suction is an important factor in bringing about a recovery. If a diseased condition of the mastoid process is evident, the desired result can naturally no longer be obtained by this method.

EPIDERMIC CYSTS, FOLLOWING TRANSPLANTATION IN THE CAVITY PRODUCED BY THE RADICAL MASTOID OPERATION.

BY DR. W. SCHOETZ, HEIDELBERG.

Translated from *Zeitsch. f. Ohrenheilk.*, Vol. LVI., No. 1, 1908, by Dr. M. J. BALLIN, New York.

IN the 39th volume of the *Archiv f. Ohrenheilk.*, Leutert describes two tumors the size of a small pea, which developed in the ear of a patient who had had a radical mastoid operation on account of the formation of cholesteatoma, four years previously, and in which the structure resembled the epidermic cysts observed by Reverdin and Garré.

Leutert states in his paper that such structures may develop, if the bone cells, in which the invading epidermis is entirely or partly preserved, become shut off secondarily by the formation of granulations at the borders, or by the transplanted epidermis. Cysts containing fluid, which are apparently lined with mucous membrane and which have taken their origin from remnants of mucous membrane which has been allowed to remain, have been repeatedly observed after radical mastoid operations.¹ A few epidermic cysts are also mentioned,² but more concise observations in reference to the formation of such cysts following the radical mastoid operation and

¹ *Sitzung der Österr. otol. Ges.* 26, iii., '06. *Monat. f. O.*, 1907, xli., 2, p. 92.

² *Sitzung der Österr. otol. Ges.* 27, x., '06. *Monat. f. O.*, 1896, xxx., 11, p. 439.

transplantation have not been reported, as far as the writer could ascertain.

Under these conditions, the following observations may be of some interest not only to the otologists, but also to surgeons and pathologists.

I. Miss O. Eighteen years old. Radical mastoid operation performed on the 2d of April, 1902, for an otitis media chronica desquamativa. A retroauricular pedunculated flap was made according to Passow,¹ which however did not take well as can be seen from the history of the case. On the 16th, transplantation of Thiersch's skin flaps into the wound. On the 9th of June, the ear was completely dry. In the beginning of April, 1905, the ear which had been operated upon began to suppurate again, and at the same time lancinating pains were present at the tip of the mastoid. As the pains disappeared after a few weeks, the patient noticed a swelling behind the ear, which induced her to return to the clinic on the 3d of May, 1905. With the exception of a small suppurative portion at the orifice of the tube, the tympanic cavity was dry and covered with epidermis. A fluctuating tumor the size of a pigeon's egg extended from the tip of the mastoid into the fossa retromandibularis. The skin was somewhat reddened and movable. There was no pain on pressure. The swelling was incised and proved to be an epidermic cyst, the contents of which was of a pulpy, sterile nature. During the extirpation, another cyst, also filled with an epidermic pulp, was found farther anteriorly. As soon as the extensive wound surface became covered with epidermis, with the exception of a small granulating surface upon which a drainage tube had been resting, the patient was discharged in the beginning of June. In the middle of May, 1907, the patient was again admitted into the clinic on account of a painful swelling behind the operated ear. From the lower border

¹ The first plastic method in the radical mastoid operation as devised by Passow (*Eine neue Transplantationsmeth. f. d. Radikaloperat. bei. chron. Mittelohreit.*, 1895), which was used in this case, has been abandoned by the writer for better methods (*Festschr. f. Lucae*, Berlin, 1905, Passow, *Gehörgansplastik bei der Radikaloperation chron. Mittelohreiterung*).

of the retroauricular opening, a linear scar ran down to the angle of the inferior maxillary bone. The cavity produced by the operation was dry and lined with healthy epidermis. Behind the uppermost portion of this scar, a tense swelling somewhat sensitive to pressure, measuring about 3cm in length and 1cm in width, and the upper white extremity of which already protruded into the cavity itself, was found beneath the slightly reddened movable skin. An incision was made around the tumor, under local anæsthesia, which was then dissected out, whereupon its contents, which was of an atheromatous nature, escaped through a small tear. Microscopic examination showed that the cyst wall was lined with squamous epithelium consisting of several layers, which rested upon a tense layer of connective tissue having no papillæ.

II.—Miss W. Sixteen years old; had a radical mastoid operation on the 30th day of April, 1907. The bone was not sclerosed, the antrum was small and filled with granulations. The plastic was made just as in the first case. The wound having pursued an uninterrupted course, the patient was discharged on the 5th of May, for ambulatory treatment. One week later, a Thiersch's skin flap taken from the arm was transplanted after the removal of unhealthy granulations, which became adherent without any difficulty. When the patient again visited the clinic at the end of October, 1907, swelling was found about the size of pigeon's egg; this nearly filled the retroauricular opening, is supposed to have developed soon after the operation, had a broad base and showed distinct fluctuation. Its contents which was yellow, like the yolk of an egg, was readily visible through the thin, easily wrinkled skin, which contained small tortuous blood-vessels. There was also a purulent discharge from the tympanic cavity. On the 29th of October the tumor was extirpated, under local anæsthesia.

After incising the skin around the cyst it was removed in toto from the smooth, bony trough to which it adhered at circumscribed parts. The wound pursuing a normal course, the patient was discharged on the 23d of November. Microscopic examination: The skin over the tumor consisted of a rather thick epidermis from which atrophic hair follicles

and some glands extended into the somewhat loosened up corium. Towards the edges of the wound it is considerably thinner; its papillæ are flat. Below the centre of the excised skin flap, the corium, which is moderately rich in round cells, is transformed into granulation tissue; this is very rich in round cells, is infiltrated with small groups of fat cells which form a portion of the cyst wall, and continues at the side in the form of a tense connective-tissue capsule poor in cells. This connective-tissue capsule, which completely surrounds the tumor, has on its inner surface a marked desquamating epidermis consisting of four to five layers of cells, the thickened borders of which seem to continue over the granulating part. The contents is formed by loose masses of scales arranged in layers like that of epidermis.

The traumatic epidermic cysts in the hands and fingers, first described by the surgeons, are supposed to be due to a cutting off of a portion of the epidermis which has been produced by a trauma of the subcutical layer. This theory can be confirmed by the fact that such cysts have been produced experimentally by Kaufmann, Ribbert, and others, and also by the post-operative development of such structures after the implantation of epidermis¹ or after incomplete excision of an ingrown nail.²

Pels-Leusden³ has recently expressed the opinion that at least a portion of traumatic epithelium cysts takes its origin from injured appendages of the skin, glands, or hair follicles. That analogous structures may also be produced by covering the epidermis with transplanted skin flaps, the writer has not found described anywhere except in the paper of Leutert.

That this possibility is emphasized by an otologist is really not surprising, as nowhere are the conditions as favorable as in an ear which has been operated upon for

¹ Worz, "Traumat. Epithelcysten," *Bruns' Beitr.*, xviii., 1897.

² Martin, "Beitr. z. Lehre von d. traum. Epithelcysten," *D. Zeitsch. f. Chir.*, xlviii., p. 597.

³ Pels-Leusden, "Traumat. Epithelcysten," *Deutsch med. Wochenschr.*, 1905, No. 34.

cholesteatoma or a desquamative otitis. Even when exercising the greatest care it may happen that, in some corner, a piece of epidermis escapes the eye of the operator, and is covered by the transplanted flap. This danger is still increased if the transplantation is undertaken immediately at the completion of the mastoid operation, as is the case in the original plastic operation of Passow; however, the delicate smooth cholesteatomatous membrane becomes separated from the bone much less readily than from the granulating surrounding area.

In the first case, in which the skin flap took poorly, it was therefore possible that, although no epidermis was allowed to remain which had been covered over, epidermis pushed its way under the flap from the surrounding skin of the neck or of the external meatus. Six weeks later Thiersch's skin flaps were transplanted, and the granulations which surrounded the skin flap were curetted; the epidermic areas, which extended somewhat under the pedunculated flap, were destroyed. These factors may perhaps most readily explain the multiple occurrence, and the peculiar long form of these cysts.

In the second case, there was no cholesteatoma, and there also seemed to be no desquamative otitis present, as the wound healed by primary union, the skin flap having taken well; and if we do not wish to assume that a partial epidermization of the mastoid cells had already existed which was overlooked at the time of the mastoid operation, the only possibility which remains is that the cyst developed from mucous membrane which later underwent a metaplasia, or from the remaining portions of epidermis. The latter supposition could not be demonstrated microscopically and the relatively loose connection of the cyst with the overlying skin, as well as its firm adhesion to circumscribed portions of the bone, speaks rather in favor of the first. That a metaplasia of the mucous membrane into an epidermic cyst occurs, is very

probable according to an observation of Brühl,¹ who found directly beneath the surface in an aural polyp, which was covered only with cylindrical epithelium and which contained cylindrical epithelial cyst, a large cavity which was lined with broad, stratified, squamous epithelium, and contained cholesteatomatous masses. According to the experiments of Ribbert² the epithelium not infrequently changes its character, if it grows in a foreign or an inflammatory area; or in other words under altered conditions necessary for its development.

The thin pulpy character of the contents of the cyst is only partly attributed to the degeneration of the epidermis generally observed in atheromata; it can, however, be partly explained by the fact that, not as in a true cholesteatoma in which a completely closed sac forms the cyst, a portion of the wall still contains granulations, as has been demonstrated at least in the second case. The contents also show that these tumors belong to the same class as the epidermic cyst first observed on the fingers, in which the fluctuating contents was mistaken for a hygroma in several cases, and distinguishes them from cholesteatomata which Borst described as a structure having a white, silk-like lustre, a dry, crumbling consistency, and a characteristic stratified leaf-like form. Leutert's tumors show on microscopic examination a greater resemblance to the true cholesteatomata, so that the original aural affection in his case may also have possibly been a true cholesteatoma, in spite of the fact that, according to the history of the case,³ no epidermis was found in the middle ear and its accessory cavities at the first operative opening up of the mastoid process.

¹ Brühl, "Beitr. z. path. Anatom. d. Gehörorg.," *Zeitschr. f. Ohrenheilk.*, 1905, xlix., p. 34.

² *Zeitschr. f. Chir.*, xlviii., p. 575.

³ *Comp. Arch. f. Ohrenheilk.*, xxxiv., p. 267.

FUNCTIONAL EXAMINATION OF THE ORGAN OF HEARING IN DEAF-MUTES.¹

A CONTRIBUTION TO THE CLINICAL PATHOLOGY OF THE
INNER EAR. (INCLUDING A TABLE OF THE
COLLECTED MATERIAL USED IN OUR
EXAMINATIONS.)

BY G. ALEXANDER AND G. W. MACKENZIE

Translated from the *Zeisch. f. Ohrenhkl.*, Vol. LVI., No. 2, 1908, by
G. W. MACKENZIE, Philadelphia.

CONSIDERABLE time has elapsed since the last thorough examinations have been made upon deaf-mutes (Bezold, Brühl, Wanner, Alexander and Kreidl, Frey and Hammerschlag, Pollak, and others), during which time our knowledge of clinical otology has been greatly increased, especially through improvements of our examination methods of the static labyrinth.

It was found necessary to examine a sufficient amount of material, adopting all of the earlier known together with the more recent methods, so far as they could be depended upon; the results of these examinations will be given later.

Our examinations have been conducted with the intention of solving some disputed questions, and especially that of **labyrinthine equilibrium disturbances**, which recently has been a subject of so much discussion. In this relation, as well as the galvanic examination methods and the examination upon the goniometer, the clini-

¹ From the Ear Department of the Allgemeinen Polyklinik in Wien.

cal examinations in the following paper have been made after the methods of G. W. Mackenzie.¹ At the present time there is considerable difference of opinion concerning the question of equilibrium disturbances; however, the existence of positive equilibrium disturbances in an appreciable number of deaf-mutes has been found uniformly by all examiners. The question though, why in some cases and not in others disturbances of equilibrium are present, appears, according to previous investigations, entirely unsolved, and the different appearing opinions in the literature are supported solely by theoretic suppositions.

According to the findings in the individual cases, we were able to group our material as follows:

I.—Deaf-mutes with complete non-reactibility of the cochlea (absolute deafness) and static labyrinth.

II.—Deaf-mutes with partial destruction of the inner ear (hearing rests) and positive irritability of the static labyrinth.

III.—Deaf-mutes with partial destruction of the inner ear (hearing rests) and complete destruction of the static labyrinth.²

IV.—Deaf-mutes with total destruction of the cochlea (absolute deafness) and positive irritability of static labyrinth.

This division permits of a better grouping of the cases than was heretofore possible. Originally the deaf-mutes were grouped solely according to the quantity of hearing rests (Itard). Later came the qualitative division with emphasis upon the hearing rests, for the various pitches of the voice (Bezold). Alexander and Kreidl, upon the basis of comparative and anatomical examination

¹ See *Arch. f. Ohrenheilk.* and *Monatschr. f. Ohrenheilk.*, 1908.

² See Neumann: "Über ein Fall zirkumskripten Labyrinthkrankung und einen Fall von Neuritis vestibularis" (Rud. Zack), Österreich. Otol. Gesellschaft, Sitzung vom Oktober, 1907, *Monatschr. f. Ohrenheilk.*

upon animals with congenital labyrinth anomalies, by applying special methods of examination, succeeded in separating congenital from acquired deafness.

By the division into congenital and acquired forms of deafness it becomes clear just where earlier mistakes had been made, because here we are too much dependent upon the history which, as we know, is very unreliable, especially in cases of early acquired deafness (deafness acquired in first or second year of life).¹

Alexander and Kreidl pointed out this unreliability, which, at the same time, led them to exclude the discussion of individual cases.

The material used in our examinations consisted of pupils of the k. k. Taubstummen Institutes in Wien, from which material earlier examinations have been made by Kreidl, Pollak, and Alexander.

To the Director of the Institute, Dr. Fink, and the Principal, Mr. Gabriel, we wish to extend our thanks for their kindness and courtesy in supplying the material and for their further assistance. The Principal, Mr. Gabriel, was kind enough to furnish us with the history and progress of the pupils in articulation, lip reading, and general capability, arranged in table form, for which we thank him in particular. The data of the individual cases comprise:

1.—Name and age.

2.—History: a brief report is given by the parents at the time of the child's admission to the institute—which, as already stated, is in many cases unreliable. We will touch upon this subject later.

¹ Hammerschlag (*Z. f. O.*, vol. 45) attempted, with success, to show the mistake of such a division by exact observation of the accompanying phenomena (Multiplizität, kongenitale Augenkrankheiten, directe und indirecte Aszendenz). In this manner he was able to establish the fact of similarity in the behavior of the vestibular apparatus of the congenitally deaf and that of dancing mice. (Compare Alexander and Kreidl, *Pflüger's Arch.*, Bd. 38).

3.—Classification noted in articulation, lip reading, and general capability.

(1—very good; 2—good; 3—sufficient; 4—barely sufficient; 5—insufficient.)

4.—Otoscopic findings.

5.—Subjective noises (ringing, hissing, etc.)

6.—Functional examination of the organ of hearing with especial attention to hearing rests.

7.—Functional examination of the non-acoustic labyrinth (static labyrinth).

(a) Spontaneous nystagmus in relation to its character (rotatory or horizontal), the direction (right, left), and intensity (apparent upon looking to the same side, straight ahead, and upon looking to the opposite side).

(b) Irritability of the static labyrinth to turning (examination upon the turning stool).

(c) Galvanic irritability recorded in milliampères, with kathode to the ear and anode to the ear.¹

8.—Equilibrium disturbances: Romberg, walking forward and backward, running forward and backward, hopping upon one foot forward and backward, all of which examinations were made with open and with closed eyes.

9.—Goniometer.² Whereby inclination is produced in the four principal directions: forward, backward, right, and left,—with open and with closed eyes. The examinations upon the goniometer were made with the patients barefooted, and in order to prevent slipping, the inclining plank of the goniometer, upon which the pupils stood, was dusted with kolophonium powder. In the table the nomenclature of Kümmel has been adopted: vh = vorn hoch (front high); hh = hinten hoch (back high); rh = rechts hoch (right high); lh = links hoch (left high).

¹ See G. W. Mackenzie concerning the "Galvanic Irritability of the Static Labyrinth." *Archiv für Ohrenheilkunde*, 1908.

² The Alexander modified Stein's goniometer was used in our examinations.

For details see G. W. Mackenzie.¹

10.—Acuity of vision.

11.—Pupillary reaction.

12.—Deep reflexes.

13.—Co-ordination.

All examinations were repeated several times in individual cases where the nature of the examination demanded it; especially so the examinations upon the goniometer.

The results of the complete examinations are summarized in the appended table.

The examination of the caloric irritability of the labyrinth was not made, for with intact membrane the examination with cold water is necessarily a slow process; the examined is frequently nauseated, and, added to this, on account of restlessness and anxiety of the patient, there is the danger of producing, with the cannula, a traumatic rupture of the membrane. In case of dry perforation it appears almost unnecessary to caution against the possibility of the recurrence of middle-ear suppuration in attempting the caloric examination. We were all the more able to exclude this examination since the examination upon the turning stool and the galvanic examination were sufficient to determine the irritability of the static labyrinth.

¹ "Klinische Untersuchungen über die labyrintharen Gleichgewichtsstörungen mit besonderer Berücksichtigung der allgemeinen Prüfungsmethoden und des Goniometers." *Monatschr. f. Ohrenheilk.*, 1908.

TABLE OF HISTORIES AND FINDINGS.

No.	Name.	Age.	History.	Articulation.	Lip Reading.	Capability.	Otoscope Findings.	Tinnitus.	Acoustic Findings.	Vestibular Apparatus.
										Spontaneous Nystagmus.
1.	Victor H.	16½	Deaf since 2d year of life after measles.	1	2	Very good.	Tympanic membranes of both sides completely destroyed. Granulations in tympanic cavities.	Negative.	Complete deafness both sides.	Very slight rotatory nystagmus to both sides when looking extremely to the sides.
2.	Norbert K.	17	Deaf since birth.	3	1	Good.	Both sides normal.	Negative.	Complete deafness both sides.	Slight rotatory nystagmus to both sides when looking extremely to the sides.
3.	Ferdinand K.	13	Greatly impaired hearing since 2d year of life. Cause unknown.	1	1	Very good.	Tympanic membrane retracted and clouded.	Negative.	Hearing greatly diminished, both sides. A ₁ (Bezold) right side much shortened, left side somewhat less shortened.	Rotatory nystagmus to both sides upon looking extremely to the sides.
4.	Anton N.	13½	Deaf since birth.	3	3	Good.	Both sides normal.	Negative.	Reaction to loud tones and noises very weak. A ₁ (Bezold) and small c ₄ both sides very much shortened.	Rotatory nystagmus to both sides upon looking extremely to the sides.
5.	Max B.	13½	Deaf since 2d year of life after blow upon the head.	1	1	Good.	Tympanic membranes of both sides thickened and clouded.	Negative.	Slight rest of hearing in both ears. A ₁ (Bezold) somewhat more shortened in the right than the left ear.	Pronounced rotatory nystagmus to the left when looking to the left, less pronounced rotatory nystagmus to left when looking straight ahead, no nystagmus to the right when looking to the right.
6.	Anton R.	15	Deaf since birth.	3	1	Good.	Both sides normal.	Negative.	Complete deafness both sides.	Slight rotatory nystagmus to both sides upon extreme looking to the sides.
7.	Rudolf E.	14	Deaf since birth.	3	2	Very good.	Tympanic membranes of both sides retracted and clouded — shadow on promontory.	Negative.	Complete deafness both sides.	Distinct rotatory nystagmus to both sides when looking to the sides; however, more distinct to the left side.
8.	Konrad K.	11½	Deaf since 5th year of life on account of a fall upon the back of the head.	1	3	Very good.	Both sides normal.	Negative.	Very marked diminution of hearing in right ear, small A ₁ (Bezold) very much shortened. Left ear complete deafness.	Rotatory nystagmus to both sides when looking to the sides; however, more marked to the right side.

TABLE OF HISTORIES AND FINDINGS

VESTIBULAR APPARATUS.		Equilibrium Disturbances.	GONIO-METER.		Vision.	Deep Reflexes.	Pupillary Reaction.	Co-ordination.
Nystagmus after Turning.	Galvanic Reaction.		Eyes open.	Eyes closed.				
After 10 turnings to both sides with head bent forward 90°, no nystagmus (reaction) to either side.	R { K 8 Ma rN A 11 Ma rN L { K 8 Ma rN A 8 Ma rN	*R Romberg negative; very broad gait with closed eyes. Hopping with open eyes fair, but with closed eyes very uncertain and pupil tends to fall.	vh 30 hh 28 rh 26 lh 27	vh 27 hh 19 rh 12 lh 11	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus (reaction) to either side.	R { K 18 Ma. No re- action. A 18 Ma rN L { K 18 Ma rN A 18 Ma. No re- action.	R Romberg positive; very uncertain and broad gait with closed eyes both when walking forward as well as backward. Hopping barely possible.	vh 28 hh 21 rh 17 lh 21	vh 19 hh 8 rh 4 lh 4	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right 4 seconds. After ten turnings to the right, with head bent forward 90°, rotatory nystagmus to the left lasting 5 seconds.	R { K 4 Ma rN A 5 Ma rN L { K 3 Ma rN A 5 Ma rN	R Romberg ±; very uncertain and broad gait with closed eyes when walking forward and backward. Hopping very uncertain.	vh 26 hh 30 rh 19 lh 28	vh 12 hh 12 rh 12 lh 18	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 16 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 16 seconds.	R { K 7 Ma rN A 6 Ma rN L { K 5 Ma rN A 9 Ma rN	R Romberg negative; gait with closed eyes forward and backward more uncertain than with eyes open. Hopping with closed eyes somewhat uncertain.	vh 30 hh 30 rh 30 lh 30	vh 25 hh 28 rh 23 lh 28	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus.	Owing to anxiety and the nervousness of the pupil it was not possible to make the examination.	R Romberg ±; uncertain, however not a broad gait with closed eyes when walking forward and backward. Hopping somewhat more uncertain with closed than with open eyes, however not very pronounced.	vh 18 hh 16 rh 19 lh 15	vh 18 hh 15 rh 12 lh 12	Not especially good on account of corneal opacities after keratitis exzematosa.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 25 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 22 seconds.	R { K 10 Ma rN A 10 Ma rN L { K 19 Ma rN A 11 Ma rN	R Romberg negative; very broad and uncertain gait. Hopping forward and backward with open and closed eyes faultless.	vh 30 hh 30 rh 30 lh 30	vh 23 hh 30 rh 30 lh 30	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus.	R { K 14 Ma. No re- action. A 14 Ma. No re- action. L { K 14 Ma. No re- action. A 14 Ma. No re- action.	R Romberg positive; very broad and uncertain gait forward and backward with closed eyes. Also hopping very uncertain.	vh 30 hh 30 rh 29 lh 30	vh 23 hh 17 rh 17 lh 11	Good.	Normal.	To light prompt.	No ataxia.
After ten turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 15 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 15 seconds.	R { K 3 Ma rN A 7 Ma rN L { K 7 Ma rN A 3 Ma rN	R Romberg negative; gait and hopping forward and backward not so certain with closed as with open eyes. However the difference is not very pronounced.	vh 30 hh 30 rh 30 lh 28	vh 25 hh 28 rh 27 lh 27	Good.	Normal.	To light prompt.	No ataxia.

*K 8 Ma rN R means Kathode 8 Milliampere rotatory Nystagmus to the Right (or to the Left) and represents the ocular movements with the examiner facing the patient.

TABLE OF HISTORIES AND FINDINGS

No.	Name.	Age.	History.	Articulation.	Lip Reading.	Capability.	Otoscope Findings.	Tinnitus.	Acoustic Findings.	VESTIBULAR APPARATUS.	
										Spontaneous Nystagmus.	
9.	Norbert H.	17	Deaf since birth.	2	1	Good.	Both sides normal.	Noises in left ear only.	Right ear complete deafness. Slight rest of hearing in left ear. Small a ₁ (Bezold) and high c ₄ very much shortened.	Pronounced rotatory nystagmus to both sides; however more marked to the right side.	After 10 left with ward 90° tagmus lasting 10 turns with h 90°, rot to the seconds.
10.	Max L.	14	Deaf since 3d year of life after scarlet fever.	2	1	Very good.	Both sides normal.	Negative.	Complete deafness both sides.	Slight rotatory nystagmus to both sides upon looking extremely to the sides.	After 1 sides forward mus.
11.	Anton N.	13½	Deaf since birth.	3	3	Good.	Both sides normal.	Negative.	Complete deafness both sides.	Very slight rotatory nystagmus to both sides when looking extremely to the sides.	After 1 left rot to the seconds ings to head 90°, rot to the seconds.
12.	Johann O.	13½	Deaf since 1st year of life after meningitis.	4	2	Good.	Both sides normal.	Negative.	Complete deafness both sides.	Slight rotatory nystagmus to both sides when looking extremely to the sides.	After 10 sides forward mus. tion.)
13.	Johann B.	13½	Deaf since 3d year of life after fall(?).	2	2	Sufficient.	Both sides normal.	Negative.	Great impairment of hearing both sides. A ₁ (Bezold) and c ₄ very much shortened.	Distinct rotatory nystagmus to both sides when looking extremely to the sides.	After 10 left with ward 90° tagmus lasting 10 turns with h 90°, rot to the le onds.
	Franz Josef T.	13½	Deaf since 6th year of life. Cause unknown.	1	1	Very good.	Membranes of both sides clouded.	Negative.	Right side slight rest of hearing. A ₁ (Bezold) very much shortened. Left side complete deafness.	Very slight rotatory nystagmus to both sides when looking extremely to the sides.	After 10 left with ward 90° tagmus ing 26 se turnings head be rotatory the left onds.
15.	Helena Z.	14	Deaf since 5th year of life after meningitis.	2	3	Sufficient.	Both sides normal.	Positive left side only.	Both sides slight rest of hearing. A ₁ (Bezold) and c ₄ very much shortened.	Very slight horizontal nystagmus of long excursions mixed with rotatory nystagmus to both sides when looking intently to the sides.	After 10 sides w forward mus. (tion.)
16.	Hermine N.	15	Deaf since 4th year of age. Cause unknown.	1	2	Good.	Both sides normal.	Positive left side only.	Hearing rests both sides. A ₁ (Bezold) and c ₄ much shortened.	Very slight rotatory nystagmus to both sides when looking extremely to the sides.	After 10 sides wit forward mus. (

TABLE OF HISTORIES AND FINDINGS

VESTIBULAR APPARATUS.		Equilibrium Disturbances.	GONIO-METER.		Vision.	Deep Reflexes.	Pupillary Reaction.	Co-ordination.
Nystagmus after Turning.	Galvanic Reaction.		Eyes Open.	Eyes Closed.				
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 33 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 27 seconds.	R { K 5 MarN A 2 MarN L { K 2 MarN A 6 MarN	R Romberg negative; gait and hopping less certain L with closed than with open eyes. Difference, however, is not pronounced.	vh 30 hh 28 rh 28 lh 28	vh 24 hh 24 rh 22 lh 22	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus.	R { K 14 Ma. No re- action. A 14 Ma. No re- action. L { K 14 Ma. No re- action. A 14 Ma. No re- action.	R Romberg positive; very uncertain and very broad gait when walking forward and backward with closed eyes; hopping not possible.	vh 28 hh 30 rh 30 lh 28	vh 22 hh 21 rh 17 lh 15	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left rotatory nystagmus to the right lasting 10 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to the left lasting 10 seconds.	R { K 18 Ma. No re- action. A 18 Ma. No re- action. L { K 10 MarN A 12 MarN	R Romberg negative; somewhat uncertain but not broad gait when walking forward and backward L with closed eyes; hopping uncertain.	vh 30 hh 29 rh 26 lh 28	vh 24 hh 26 rh 21 lh 17	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus. (Negative reaction.)	R { K 5 MarN A 7 MarN L { K 7 MarN A 7 MarN	R Romberg positive; very broad gait and very uncertain when walking forward and backward with closed eyes; hopping very uncertain.	vh 27 hh 28 rh 27 lh 26	vh 20 hh 28 rh 22 lh 16	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 12 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 10 seconds.	R { K 14 Ma. No re- action. A 10 MarN L { K 12 MarN A 12 Ma. No re- action.	R Romberg negative; very broad gait and very uncertain when walking forward and backward with closed eyes; hopping very uncertain.	vh 28 hh 28 rh 28 lh 28	vh 22 hh 27 rh 22 lh 27	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 26 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 25 seconds.	R { K 10 MarN A 10 MarN L { K 10 MarN A 10 MarN	R Romberg negative; gait and hopping forward and backward with closed eyes almost as well as with open eyes.	vh 30 hh 30 rh 30 lh 30	vh 28 hh 29 rh 30 lh 28	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus. (Negative reaction.)	R { K 15 Ma. No re- action. A 11 MarN L { K 4 MarN A 15 Ma. No re- action.	R Romberg negative; uncertain and broad gait when walking forward and backward; hopping scarcely able.	vh 28 hh 25 rh 25 lh 22	vh 10 hh 17 rh 16 lh 15	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head inclined forward 90°, no nystagmus. (Neg. reaction.)	R { K 12 Ma. No re- action. A 12 Ma. No re- action. L { K 12 Ma. No re- action. A 12 Ma. No re- action.	R Romberg positive. Very uncertain and very broad gait when walking forward and backward with closed eyes. Hopping not possible.	vh 24 hh 28 rh 11 lh 24	vh 14 hh 14 rh 8 lh 4	Good.	Normal.	To light prompt.	No ataxia.

TABLE OF HISTORIES AND FINDINGS

No.	Name.	Age.	History.	Articulation.		Capability.	Otoscope Findings.	Tinnitus.	Acoustic Findings.	Vestibular Apparatus.
										Spontaneous Nystagmus.
17.	Marie St.	14½	Deaf since 2d year of life through fall from baby carriage.	3	3	Good.	Both sides normal.	Positive left side only.	Right side complete deafness. Left side rest of hearing. A ₁ (Bezold) and c ₄ heard when struck very strong blow with rubber hammer.	Distinct rotatory nystagmus to both sides when looking extremely to the sides.
18.	Olga F.	12½	Deaf since birth.	3	2	Good.	Both sides normal.	Negative.	Complete deafness both sides.	Very slight rotatory nystagmus to both sides when looking extremely to the sides.
19.	Karl B.	11½	Deaf since 11th year of age after scarlet fever.	1	2	Good.	Both sides normal.	Negative.	Complete deafness both sides.	Rotatory nystagmus to both sides when looking extremely to the sides.
20.	Aloisia F.	15½	Deaf since 5th year of age after meningitis.	2	1	Good.	Membrane retracted both sides.	Positive left side only.	Complete deafness both sides.	Pronounced horizontal nystagmus to both sides when looking intently to the sides, and slight rotatory nystagmus to the right when looking straight ahead.
21.	Mathilde W.	15½	Deaf since birth.	2	2	Good.	Membrane normal left side, slightly clouded right side.	Positive both sides.	Slight hearing rest both sides. A ₁ (Bezold) and c ₄ very much shortened.	Slight rotatory nystagmus to both sides when looking extremely to the sides.
22.	Olga W. (Sister of No. 21.)	14	Deaf since birth.	3	3	Sufficient.	Both membranes normal.	Positive right side only.	Complete deafness both sides.	Very slight rotatory nystagmus to both sides when looking extremely to the sides.
23.	Marie B.	14½	Deaf since 3d year of life after meningitis.	1	1	Very good.	Both membranes normal.	Positive left side only.	Complete deafness both sides.	Pronounced rotatory nystagmus to the right when looking intently to the right, but no nystagmus to the left when looking to the left.

TABLE OF HISTORIES AND FINDINGS

VESTIBULAR APPARATUS.		Equilibrium Disturbances.	GONIO-METER.		Vision.	Deep Reflexes.	Pupillary Reaction.	Co-ordination.
Nystagmus after Turning.	Galvanic Reaction.		Eyes Open.	Eyes Closed.				
After 10 turnings to left with head bent forward 90°, rotatory nystagmus to the right lasting 11 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 17 seconds.	R { K 10 MarN A 10 MarN L { K 8 MarN A 11 MarN	R Romberg positive. Gait L and hopping forward and L backward with closed eyes R almost as well as with open eyes.	vh 22 hh 27 rh 28 lh 28	vh 20 hh 26 rh 21 lh 24	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 17 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 20 seconds.	R { K 5 MarN A 4 MarN L { K 4 MarN A 5 MarN	R Romberg negative. Gait L and hopping forward and L backward with closed eyes R almost as well as with open eyes.	vh 26 hh 29 rh 29 lh 29	vh 18 hh 23 rh 29 lh 29	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 15 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 8 seconds.	R { K 3 MarN A 3 MarN L { K 3 MarN A 3 MarN	R Romberg negative somewhat broad gait with closed eyes, pupil takes short steps. Hopping with closed eyes tolerably good.	vh 30 hh 30 rh 28 lh 30	vh 25 hh 23 rh 22 lh 22	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 6 seconds. After 10 turnings to the right with head bent forward 90°, no nystagmus. (Neg. reaction from left side.)	R { K 3 MarN increased. A 16 Ma No reaction. L { K 12 Ma No reaction. A 12 Ma No reaction.	R Romberg negative. Very uncertain gait when walking forward and backward with closed eyes. Hopping very uncertain. No dizziness.	vh 13 hh 30 rh 28 lh 30	vh 14 hh 11 rh 21 lh 11	Fair.	Normal.	To light prompt.	
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 20 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 20 seconds.	R { K 10 MarN A 5 MarN L { K 6 MarN A 10 MarN	R Romberg negative. Gait L and hopping forward and L backward with closed eyes R as steady as with open eyes.	vh 28 hh 27 rh 28 lh 28	vh 23 hh 27 rh 17 lh 28	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 14 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 16 seconds.	R { K 6 MarN A 6 MarN L { K 8 MarN A 6 MarN	R Romberg negative. Gait L and hopping when walking forward and backward with closed eyes almost as steady as with open eyes.	vh 29 hh 29 rh 28 lh 28	vh 23 hh 28 rh 24 lh 25	Not so good on account of uncorrected high myopia.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus. (Neg. reaction.)	R { K 14 Ma. No reaction. A 14 Ma. No reaction. L { K 14 Ma. No reaction. A 14 Ma. No reaction.	R Romberg negative. Uncertain gait when walking forward and backward (with short steps) with closed eyes. Hopping very poor.	vh 29 hh 27 rh 26 lh 17	vh 22 hh 22 rh 18 lh 18	Left eye not so good on account of abducens paralysis, etc. Right eye vision good.	Normal.	To light prompt.	No ataxia.

TABLE OF HISTORIES AND FINDINGS

No.	Name.	Age.	History.	Articulation.	Lip Reading.	Capability.	Otoscope Findings.	Tinnitus.	Acoustic Findings.	VESTIBULAR APPARATUS.	
										Spontaneous Nystagmus.	
24.	Marie G.	15	Deaf since 3d year of life after a fall.	1	1	Sufficient.	Both membranes normal.	Positive right side only.	Right side slight hearing rest present. C ₄ very short. Left side complete deafness.	Slight rotatory nystagmus to both sides when looking extremely to the sides.	
25.	Aloisia S.	14	Deaf since birth.	4	1	Very good.	Both membranes normal.	Positive both sides.	Slight hearing rest both sides, small a ₁ (Bezold) not heard upon either side. c ₄ very short.	Rotatory nystagmus to both sides when looking extremely to the sides.	
26.	Emilie S. (Sister to No. 25.)	12	Deaf since birth.	3	2	Sufficient.	Double-sided chronic adhesive process with retracted membranes.	Negative.	Complete deafness both sides.	Rotatory nystagmus to both sides when looking extremely to the sides.	
27.	Angela W.	13½	Deaf since 6th year of age after measles.	1	1	Very good.	Membrane both sides completely destroyed.	Positive left side only.	Complete deafness both sides.	Slight rotatory nystagmus to both sides when looking extremely to the sides.	
28.	Alfred S.	23	Deaf since 20th year after scarlet fever.	1	1	Very good.	Membranes both sides retracted and clouded.	Positive both sides.	Complete deafness both sides.	Marked rotatory nystagmus to both sides upon looking extremely to the sides.	
29.	Leopoldine K.	9	Deaf since July, 1907, after meningitis.	1	3	Very good.	Membrane left side normal, right side slightly clouded.	Positive both sides.	Complete deafness both sides.	Slight rotatory nystagmus to both sides when looking extremely to the sides.	
30.	Ferdinand L.	10	Hard of hearing since birth.	1	1	Good.	Membrane both sides retracted and clouded.	Negative.	Slight rest of hearing both sides, a ₁ and c ₄ much shortened.	Very slight rotatory nystagmus to both sides when looking extremely to the sides.	
31.	Wilhelm W.	11 J.	Deaf after whooping-cough in 8th year.	3	3	Sufficient.	Membrane right side somewhat clouded with chalk deposits. Left membrane clouded and retracted.	Negative.	Left side slight rest of hearing, small a ₁ and c ₄ very much shortened. Right side completely deaf.	Rotatory nystagmus to both sides in looking extremely to the sides. More pronounced to the right than the left.	

TABLE OF HISTORIES AND FINDINGS

VESTIBULAR APPARATUS.		Equilibrium Disturbances.	GONIO-METER.		Vision.	Deep Reflexes.	Pupillary Reaction.	Co-ordination.
Nystagmus after Turning.	Galvanic Reaction.		Eyes Open.	Eyes Closed.				
After 10 turnings to both sides with head bent forward 90°, no nystagmus. (Negative reaction.)	R { K 4 MarN A 12 MarN L { K 8 MarN A 4 MarN	R Romberg positive; gait L and hopping with closed L eye both forward and R backward very uncertain with falling to the sides.	vh 23 hh 28 rh 23 lh 24	vh 22 hh 22 rh 14 lh 18	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 16 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 4 seconds.	R { K 1 MarN A 4 MarN L { K 3 MarN A 1 MarN	R Romberg negative; gait L and hopping with closed L eyes somewhat uncertain, R with tendency to fall laterally.	vh 27 hh 14 rh 16 lh 19	vh 22 hh 12 rh 16 lh 9	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 6 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 4 seconds.	R { K 2 MarN A 2 MarN L { K 3 MarN A 2 MarN	R Romberg positive; gait L when walking forward L and backward very unsteady and broad with closed eyes; hopping scarcely possible.	vh 21 hh 22 rh 15 lh 21	vh 18 hh 15 rh 8 lh 17	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 17 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 14 seconds.	R { K 10 MarN A 7 MarN L { K 7 MarN A 10 MarN	R Romberg negative; gait L and hopping forward and backward with closed eyes R almost as well as with open eyes.	vh 29 hh 30 rh 24 lh 30	vh 24 hh 30 rh 19 lh 28	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus. (Negative reaction.)	R { K 7 MarN A 10 MarN L { K 7 MarN A 7 MarN	R Romberg positive; gait L forward and backward L very unsteady and broad with closed eyes; hopping scarcely possible.	vh 26 hh 29 rh 26 lh 30	vh 17 hh 13 rh 10 lh 11	Both sides diminished on account of recent keratitis interstitialis luetica hereditaria.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus. (Negative reaction.)	R { K 12 Ma. No re- action. A 12 Ma. No re- action. L { K 12 Ma. No re- action. A 12 Ma. No re- action.	R Romberg positive; gait with closed eyes very uncertain; hopping almost or quite impossible with closed eyes.	vh 21 hh 23 rh 15 lh 20	vh 12 hh 12 rh 7 lh 9	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 16 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 22 seconds.	R { K 4 MarN A 3 MarN L { K 2 MarN A 4 MarN	R Romberg negative; gait L and hopping forward and backward with closed eyes R almost as well as with open eyes.	vh 27 hh 30 rh 26 lh 27	vh 22 hh 26 rh 19 lh 21	Good.	Normal.	To light prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus.	R { K 10 Ma. No re- action. A 8 Ma L { K 10 Ma. No re- action. A 10 Ma. No re- action.	R Romberg positive; gait forward and backward with closed eyes very uncertain and broad; hopping barely possible.	vh 25 hh 27 rh 16 lh 24	vh 13 hh 10 rh 10 lh 10	Good.	Normal.	Prompt.	No ataxia.

TABLE OF HISTORIES AND FINDINGS

No.	Name.	Age.	History.	Articulation.	Lip Reading.	Capability.	Otoscope Findings.	Tinnitus.	Acoustic Findings.	Vestibular Apparatus.
										Spontaneous Nystagmus.
32.	Adolf K.	13½	Deafness since birth the result of forceps delivery.	2	2	Sufficient.	Both membranes retracted and clouded.	Negative.	Left side slight rest of hearing. A ₁ and c ₄ shortened. Right side completely deaf.	Horizontal nystagmus to both sides upon looking to the extreme sides.
33.	Heinr. Baron M.	12	Deaf since birth. (Parents also deaf.)	3	3	Sufficient.	Both sides normal.	Negative.	Left side slight rest of hearing. A ₁ not heard and c ₄ only by strongest irritation. Right side completely deaf.	Slight rotatory nystagmus to both sides upon looking to the extreme sides.
34.	Aloisia St.	14½	Deaf since birth.	4	2	Very good.	Membrane both sides clouded.	Positive upon left side.	Complete deafness both sides.	Slight rotatory nystagmus to both sides upon looking to the extreme sides.
35.	Fredericke F.	12	Deaf since birth.	3	2	Good.	Both sides normal.	Negative.	Complete deafness both sides.	No nystagmus to either side upon looking to the extreme sides.
36.	Stephanie P.	12½	Deaf after meningitis in 2d year. (Syndactylie of left extremity.)	2	1	Very good.	Tympanic membrane both sides clouded.	Negative.	Complete deafness both sides.	No nystagmus to either side when looking intently to the sides.
37.	Josefine P.	13	Deaf in 2d year after teething and convulsions.	2	1	Sufficient.	Tympanic membrane both sides retracted and clouded.	Positive both sides.	Slight rest of hearing both sides. A ₁ (Bezold) and c ₄ shortened.	Very slight rotatory nystagmus to both sides when looking extremely to the sides.
38.	Theresia W.	13½	Deaf after convulsions in first year.	2	2	Sufficient.	Both membranes clouded.	Positive left side only.	Left side slight rest of hearing. A ₁ and c ₄ shortened. Right side complete deafness.	Marked rotatory nystagmus to the right when looking to the right less to the left when looking to the left.

TABLE OF HISTORIES AND FINDINGS

VESTIBULAR APPARATUS.		Equilibrium Disturbances.	GONIO-METER.		Vision.	Deep Reflexes.	Pupillary Reaction.	Co-ordination.
Nystagmus after Turning.	Galvanic Reaction.		Eyes Open.	Eyes Closed.				
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 14 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to the left lasting 16 seconds.	R } K 4 MarN A 3 MarN L } K 3 MarN A 5 MarN	R Romberg negative; gait forward and backward L with closed eyes somewhat uncertain; hopping somewhat uncertain.	vh 26 hh 25 rh 30 lh 25	vh 17 hh 18 rh 17 lh 15	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 16 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 15 seconds.	R } K 4 MarN A 4 MarN L } K 4 MarN A 4 MarN	R Romberg negative; gait and hopping somewhat more uncertain with closed eyes than with open eyes. (This child was rather clumsy.)	vh 22 hh 25 rh 26 lh 25	vh 11 hh 27 rh 22 lh 10	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 16 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 19 seconds.	R } K 5 MarN A 12 Ma. No reaction. L } K 12 Ma. No reaction. A 4 MarN	R Romberg negative; somewhat more uncertain in gait and hopping with closed than with open eyes. (This child like the preceding child was normally clumsy.)	vh 21 hh 30 rh 30 lh 27	vh 18 hh 30 rh 16 lh 15	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 21 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to the left lasting 20 seconds. le	R } K 4 MarN A 10 MarN L } K 10 MarN A 6 MarN	R Romberg negative; gait and hopping more uncertain with closed than with open eyes; gait, however, was not broad.	vh 25 hh 30 rh 30 lh 30	vh 19 hh 29 rh 27 lh 29	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to both sides with head inclined forward 90°, no nystagmus. (Negative reaction.)	R } K 12 Ma. No reaction. A 12 Ma. No reaction. L } K 12 Ma. No reaction. A 12 Ma. No reaction.	R Romberg ±; very uncertain and broad gait when walking forward and backward with closed eyes; hopping impossible with closed eyes.	vh 30 hh 30 rh 26 lh 30	vh 19 hh 21 rh 14 lh 17	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 17 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to the left lasting 20 seconds.	R } K 8 MarN A 2 MarN L } K 2 MarN A 6 MarN	R Romberg negative; gait and hopping with closed eyes somewhat more uncertain with closed than with open eyes.	vh 15 hh 22 rh 23 lh 19	vh 16 hh 22 rh 14 lh 16	Right eye vision poor on account of macula cornea and convergence. Right eye. Left eye (fixing eye) vision good.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 12 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to the left lasting 13 seconds.	R } K 6 MarN A 8 MarN L } K 6 MarN A 10 MarN	R Romberg negative; swaying when walking forward and backward with closed eyes; but gait is not broad; hopping with closed eyes rather poor.	vh 18 hh 21 rh 24 lh 19	vh 16 hh 22 rh 14 lh 14	Good.	Normal.	Prompt.	Patient is somewhat clumsy but no ataxia.

TABLE OF HISTORIES AND FINDINGS

No.	Name.	Age.	History.	Articulation.	Lip Reading.	Capability.	Otoscope Findings.	Tinnitus.	Acoustic Findings.	VESTIBULAR APPARATUS.
										Spontaneous Nystagmus.
39.	Aloisa L.	10½	Hardness of hearing; cause and time of occurrence not ascertainable.	1	1	Good.	Both membranes slightly retracted.	Negative.	Slight rest of hearing both sides. A ₁ and c ₄ very much shortened.	Rotatory nystagmus to both sides when looking extremely to the sides.
40.	Frans L. (Brother to No. 39.)	18	Deaf since birth.	2	1	Good.	Both membranes normal.	Negative.	Slight rests of hearing both sides. A ₁ and c ₄ shortened more on right than left side.	Slight rotatory nystagmus to both sides when looking extremely to the sides.
41.	Christine G.	10	Deaf since birth.	2	1	Good.	Both membranes clouded.	Negative.	Complete deafness both sides.	Pronounced rotatory nystagmus to both sides when looking extremely to the sides.
42.	Philomene F.	10	Deaf after measles in 2d year.	1	1	Good.	Both membranes retracted and promontory shows red.	Positive right side only.	Slight rest of hearing both sides. A ₁ (Bezold) very short, c ₄ not heard.	No nystagmus to either side when looking extremely to the sides.
43.	Marie P.	12	Deaf after measles in 4th year.	1	1	Good.	Otitis med. sup. chron. dextra, with total destruction of the membrane; left side atresia of the meatus, post operative.	Positive, very slight left side.	Hears only very loudest tones. A ₁ and c ₄ shortened.	Horizontal and rotatory nystagmus to both sides when looking to the sides.
44.	Margaret W.	9½	Deaf after meningitis, time not ascertainable.	1	1	Very good.	Normal.	Negative.	Slightest rest of hearing. A ₁ (Bezold) very short, c ₄ not heard.	Rotatory nystagmus to both sides when looking to the sides.
45.	Mano G.	22	Deaf after meningitis in 12th year.	1	1	Very good.	Normal.	Positive both sides; stronger upon left side.	Complete deafness both sides.	Slight rotatory nystagmus to both sides when looking extremely to the sides.
46.	Eduard U.	16	Deaf after typhoid fever in 3rd year.	5	3	Very good.	Both sides normal.	Negative.	Complete deafness both sides.	Very slight rotatory nystagmus to both sides when looking to the sides.

TABLE OF HISTORIES AND FINDINGS

VESTIBULAR APPARATUS		Equilibrium Disturbances.	GONIO-METER		Vision.	Deep Reflexes.	Pupillary Reaction.	Coordination.
Nystagmus after Turning.	Galvanic Reaction.		Eyes Open.	Eyes Closed.				
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 15 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to left lasting 15 seconds.	R { K 6 MarN A 4 MarN L { K 4 MarN A 6 MarN	R Romberg negative. Gait slightly broad and somewhat uncertain when walking backward. Hopping not so bad.	vh 28 hh 28 rh 26 lh 28	vh 15 hh 28 rh 11 lh 16	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 24 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to the left lasting 22 seconds.	R { K 5 MarN A 8 MarN L { K 8 MarN A 4 MarN	R Romberg negative. Gait and hopping with closed eyes quite as good as with open eyes.	vh 29 hh 28 rh 29 lh 29	vh 24 hh 26 rh 29 lh 23	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 11 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to the left lasting 9 seconds.	R { K 4 MarN A 4 MarN L { K 4 MarN	R Romberg negative. Gait and hopping forward and backward with closed eyes quite as good as with open eyes.	vh 28 hh 28 rh 26 lh 23	vh 11 hh 27 rh 24 lh 13	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with head inclined 90° forward, rotatory nystagmus to the right lasting 9 seconds. After 10 turnings to the right with head inclined 90° forward, rotatory nystagmus to the left lasting 12 seconds.	R { K 6 MarN A 4 MarN L { K 6 MarN A 4 MarN	R Romberg negative. Gait forward and backward with closed eyes almost as good as with open eyes. Hopping the same.	vh 27 hh 27 rh 27 lh 28	vh 27 hh 26 rh 26 lh 28	Right eye blind with ant. synechia and bulb atrophic, strabismus converg. Left eye normal.	Normal.	Prompt in left eye.	Patient somewhat clumsy; however, no distinct ataxia.
After 10 turnings to the left with head inclined forward 90°, rotatory nystagmus to the right lasting 19 seconds. After 10 turnings to the right with head inclined forward 90°, rotatory nystagmus to the left lasting 19 seconds.	R { K 6 MarN A 4 MarN L { K 4 MarN A 6 MarN	R Romberg positive. Gait forward and backward with closed eyes broad and with short steps. Hopping somewhat uncertain.	vh 25 hh 24 rh 22 lh 25	vh 23 hh 25 rh 20 lh 23	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to both sides with head inclined forward 90°, no nystagmus. (Neg. reaction.)	R { K 12 Ma. No reaction. A 12 Ma. No reaction. L { K 12 Ma. No reaction. A 12 Ma. No reaction.	R Romberg positive. Gait forward and backward with closed eyes very uncertain and broad. Hopping impossible with closed eyes.	vh 19 hh 22 rh 17 lh 17	vh 14 hh 13 rh 7 lh 7	Good.	Normal.	Prompt.	Patient clumsy but no ataxia.
After 10 turnings to both sides with head inclined forward 90°, no nystagmus. (Negative reaction.)	R { K 10 MarN A 12 MarN L { K 12 MarN A 6 MarN	R Romberg ±. Gait forward and backward with closed eyes very uncertain and broad. Hopping very poor with closed eyes.	vh 30 hh 29 rh 30 lh 23	vh 15 hh 11 rh 8 lh 9	Vision fair in spite of slight secondary atrophy of both papillae.	Normal.	Right pupil does not react so promptly as left.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus.	R { K 12 Ma. No reaction. A 12 Ma. No reaction. L { K 12 Ma. No reaction. A 12 Ma. No reaction.	R Romberg negative. Gait when walking forward and backward with closed eyes uncertain and swaying. Hopping impossible.	vh 30 hh 30 rh 30 lh 30	vh 16 hh 14 rh 7 lh 7	Good.	Normal.	Prompt.	Patient somewhat clumsy; however, no distinct ataxia.

TABLE OF HISTORIES AND FINDINGS

No.	Name.	Age.	History.	Articulation.	Lip Reading.	Capability.	Otoscope Findings.	Tinnitus.	Acoustic Findings.	VESTIBULAR APPARATUS.
										Spontaneous Nystagmus.
47.	Georg Sp.	11	Deaf of unknown origin and time.	5	5	Good.	Right side membrane slightly retracted and atrophic. Left side normal.	Negative.	Slight rests of hearing both sides. A ₁ (Bezold) shortened, c ₄ not heard.	Slight rotatory nystagmus to both sides when looking to the sides.
48.	Alfred E.	11	Deaf since birth.	2	2	Sufficient.	Both sides normal.	Negative.	Slight rests of hearing present both sides. C ₄ not heard and a ₁ (Bezold) much shortened.	Rotatory nystagmus to both sides when looking to the sides; however more marked to the right.
49.	Leonhardus A.	11	Deaf in 1st year from a fall.	1	1	Very good.	Both sides normal.	Negative.	Slight rests of hearing both sides. A ₁ (Bezold) and c ₄ shortened.	No rotatory nystagmus to both sides when looking to the sides.
50.	Gustav S.	10	Deaf since 5th year after a fever of long duration.	2	1	Sufficient.	Both sides normal.	Negative.	Complete deafness both sides.	Rotatory nystagmus to right side when looking to right, no nystagmus to left side.
51.	Franz L.	10	Deaf since 8th year from a fall on the back of the head.	1	3		Both membranes slightly retracted and clouded.	Negative.	Complete deafness both sides.	Slight rotatory nystagmus to both sides when looking to the sides.

TABLE OF HISTORIES AND FINDINGS

VESTIBULAR APPARATUS.		Equilibrium Disturbances.	GONIO-METER.		Vision.	Deep Reflexes.	Pupillary Reaction.	Co-ordination.
Nystagmus after Turning.	Galvanic Reaction.		Eyes Open.	Eyes Closed.				
After 10 turnings to the left with head bent forward 90°, rotatory nystagmus to the right lasting 20 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 18 seconds.	R { K 8 Ma rN A 12 Ma rN L { K 12 Ma rN A 8 Ma rN	R Romberg negative; gait and hopping forward and backward with closed eyes somewhat more uncertain than with open eyes.	vh 26 hh 30 rh 28 lh 27	vh 19 hh 25 rh 13 lh 19	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus.	R { K 12 Ma. No reaction. A 12 Ma. No reaction. L { K 12 Ma. No reaction. A 12 Ma. No reaction.	R Romberg positive; gait forward and backward with closed eyes very uncertain, swaying and broad; hopping hardly possible.	vh 27 hh 30 rh 26 lh 27	vh 17 hh 21 rh 10 lh 10	Good (recent conjunctivitis eczematosa). 1st attack.	Normal.	Prompt.	No ataxia.
After 10 turnings to the left with the head bent forward 90°, rotatory nystagmus to the right lasting 8 seconds. After 10 turnings to the right with head bent forward 90°, rotatory nystagmus to the left lasting 9 seconds.	R { K 10 Ma. No reaction. A 10 Ma. No reaction. L { K 10 Ma. No reaction. A 10 Ma. No reaction.	R Romberg negative; gait and hopping forward and backward with closed eyes almost as well as with open eyes.	vh 26 hh 30 rh 29 lh 29	vh 28 hh 28 rh 27 lh 25	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus.	R { K 12 Ma. No reaction. A 12 Ma. No reaction. L { K 12 Ma. No reaction. A 12 Ma. No reaction.	R Romberg ±; gait forward and backward with closed eyes uncertain and broad; hopping hardly possible.	vh 27 hh 30 rh 28 lh 22	vh 21 hh 19 rh 14 lh 9	Good.	Normal.	Prompt.	No ataxia.
After 10 turnings to both sides with head bent forward 90°, no nystagmus.	R { K 10 Ma. No reaction. A 10 Ma. No reaction. L { K 10 Ma. No reaction. A 10 Ma. No reaction.	R Romberg positive; gait with closed eyes forward and backward swaying and broad, hopping impossible.	vh 25 hh 28 rh 22 lh 25	vh 6 hh 11 rh 3 lh 5	Good.	Normal.	Prompt.	No ataxia.

RESULTS.

The **Cause** of the deafness.—Of the 51 deaf-mutes examined, 49 are at the present time pupils of the k. k. Deaf-Mute Institute in Wien. Two, Alfred Slawik (No. 28) and Mano Graf (No. 45), are graduates of the same institution. The 49 examined pupils proved to be apt subjects for examination, while a great number of less intelligent pupils were not drawn upon.

The histories showed the cause of deafness to be as follows:

Congenital deafness: 18 cases (Nos. 2, 4, 6, 7, 9, 11, 18, 21, 22, 25, 26, 30, 32, 33, 34, 35, 41, 48).

Acquired deafness: 33 cases, which include meningitis 8 cases (Nos. 12, 15, 20, 23, 29, 36, 44, 45). To these may be added 2 cases (Nos. 37 and 38) which, though the ascribed cause of deafness was teething and convulsions, etiologically may be better classified under that of meningitis.

Injury (fall upon the head in early childhood): 7 cases (Nos. 5, 8, 13, 17, 24, 49, 51).

Measles: 4 cases (Nos. 1, 27, 42, 43).

Scarlet-fever: 3 cases (Nos. 10, 19, 28).

Whooping-cough: 1 case (No. 31).

Typhoid: 1 case (No. 46).

Cause of deafness unknown: five cases (Nos. 3, 14, 16, 39, 47).

According to the age at which appearance of deafness occurred the histories show:

Occurrence of deafness in	1st year,	4 cases	(Nos. 12, 38, 42, 49).
" " " "	2d " "	6 " "	" 1, 3, 5, 17, 36, 37).
" " " "	3d " "	5 " "	" 10, 13, 23, 24, 46).
" " " "	4th " "	2 " "	" 16, 43).
" " " "	5th " "	4 " "	" 8, 15, 20, 50).
" " " "	6th " "	2 " "	" 14, 27).
" " " "	8th " "	3 " "	" 29, 31, 51).
" " " "	11th " "	1 case	(No. 19).
" " " "	12th " "	1 " "	" 45).
" " " "	20th " "	1 " "	" 28).

The histories appear unreliable in the following cases:

Case 5 (deafness occurring in the 2d year through a fall upon the head) is probably a meningitis-deafness; likewise Cases 17 and 49. Therefore, the above classification should be so revised as to include under meningitis-deafness 13 cases, leaving but 4 cases of traumatic deafness.

In reference to the **articulation**, one is impressed with the fact that the males, as well as the females, with acquired deafness, articulated much better than those with congenital deafness. In all, 19 pupils showed articulation 1 = very good; of these, 10 were males and 9 were females. The 10 male pupils include 9 cases of acquired and 1 case of congenital deafness; the 9 female pupils include 8 cases of acquired and 1 case of congenital deafness. It was demonstrated, though long known, that the articulation was better the later the deafness was acquired. From these figures we further recognize the importance of hearing for the learning of articulation. A child who had heard at all after birth (even though the deafness appeared in the earliest years of life) learns to articulate much better than though the child had been deaf from birth.

On the contrary, it appears that rests of hearing are of no special benefit for the articulation.

Among 19 of the best pupils, 12 showed hearing rests, while 7 were totally deaf.

The possible association of proficiency of articulation to deafness through meningitis is shown by the following:

Among the 10 cases of meningitis-deafness, four showed under articulation progress 1, five showed progress 2, and one showed progress 4.

Since in cases of meningitis-deafness, remaining pathologic changes in the brain are quite possible, it is not surprising if they articulate poorly. This is not, however, the case. From the articulation, we are unable to obtain any proof that in cases of deafness after meningitis any special cortical changes remain. We shall refer to this

point later when we come to the discussion of labyrinthine equilibrium disturbances.

Under **capability** (general mental development) as furnished by the teachers of the institute, 17 pupils are rated "very good," 19 "good," and 12 "sufficient." These figures are of value to us. There was not one among them whose intelligence was not sufficient to permit of a satisfactory examination.

Otoscopic findings: In 26 cases the tympanic membrane was normal. In only three cases was there a double-sided chronic middle-ear suppuration with perforation or complete destruction of the membrane. The remaining cases showed changes in the form of clouding, retraction, or other catarrhal changes of the membrane.

Tinnitus or subjective noises in one or both sides were present in 20 cases; total deafness, 20 cases; hearing rests, 27 cases. Of the cases with hearing rests 9 were of congenital and 18 of acquired deafness; of the totally deaf, 10 were of congenital and 14 of acquired deafness.

Among the cases with subjective noises were 11 with hearing rests (Nos. 9, 15, 16, 17, 21, 24, 25, 37, 38, 42, and 43), and 9 totally deaf (Nos. 20, 22, 23, 27, 28, 29, 35, 45, and 51). It is of interest to note that among these totally deaf, who complain of subjective noises, we should find two of congenital deafness (Nos. 22 and 35).¹

Static labyrinth: Of the 51 examined pupils, 21 showed double-sided absolute non-irritability of the static labyrinth, although only 4 of these showed no form of spontaneous nystagmus even in extreme lateral position of the eyeballs. Of the remaining 17 cases of double-sided labyrinth destruction, we were unable to accept the spontaneous nystagmus as labyrinthine. Evidently in this case the nystagmus was produced by the unusual strain upon the eye muscles (in the extreme lateral position). This fact forces upon our attention the neces-

¹ See Alice V. Mackenzie: "Zur Klinik der galvanischen Akustikusreaktion." *Wiener klin. Wochenschr.*, März, 1908.

sity of differentiation of labyrinthine nystagmus from all other forms of nystagmus in our examination of cases without deafness.

As stated above, 21 cases showed absolute non-irritability of the static labyrinth (**no reaction after turning; with great diminution of, or negative, galvanic reaction**). While 21 cases showed complete destruction of static labyrinth, 24 showed complete destruction of acoustic labyrinth. A comparison of these figures shows that the acoustic apparatus and its nerve is somewhat more sensitive to noxious influences than the static labyrinth and its nerve; however, the difference is not great. From the results of former investigations, we expected to find a much greater difference.

Wanner (1902) found, first, that deaf-mutes who show no nystagmus after turning, at the same time show no equilibrium disturbance (Schwanken); on the contrary, those who show nystagmus after turning, at the same time show positive equilibrium disturbance. The cases of the first group comprise those of destruction; while cases of the second group comprise those of more or less intact vestibular apparatus. Wanner found in his material (108 deaf-mute pupils) no nystagmus after turning in 34.7%, a figure which agrees approximately with ours. Bezold (1898) found in his examinations 28.8%. Denker, who limited his examinations to those with complete deafness, found 42.9% showing no nystagmus after turning. That Denker's figures should exceed those of Bezold, Wanner, and ours, is readily explained when we recall that he (Denker) included in his examinations cases only of complete deafness, while the other authors included pupils with hearing rests and those without (totally deaf).

Eight cases (Nos. 6, 19, 22, 26, 27, 34, 35, 41) showed **complete deafness** with remaining (of course, not normal) irritability of the static labyrinth. Six cases (Nos. 5, 15, 24, 31, 44, 48) showed **hearing rests** with **negative irritability of the labyrinth** and, too, all these cases

showed positive equilibrium disturbances. Fourteen cases (Nos. 1, 2, 7, 10, 12, 18, 23, 28, 29, 36, 45, 50, 51) showed **complete deafness** and **absolute non-irritability** of the **static labyrinth**, all of which cases showed in addition most pronounced equilibrium disturbances. Among these 14 cases, 5 were deafness after meningitis, 3 were congenital deafness, 1 was deafness after trauma, 2 were deafness after scarlet-fever, 1 after measles, 1 after long-continued fever (probably meningitis), and finally 1 after typhoid fever. We have been able to group the congenital and acquired form of deafness, according to the reactions, into the following:

TABLE I.

	Total Number.	Congenital Deafness.	Acquired Deafness.
I.—Total deafness with negative reactivity of the static labyrinth	15	3	12
II.—Hearing rests with positive reactivity of the static labyrinth	20	7	13
III.—Total deafness with positive reactivity of the static labyrinth	9	7	2
IV.—Hearing rests with negative reactivity of the static labyrinth	7	1	6
Total	51	18	33

According to the above table, the figures prove that the great majority of cases show similar changes in both functions; in form I., where both acoustic and static labyrinth were completely destroyed, and in form II., where both acoustic and static labyrinth showed rests of functions. In the two forms, I. and II., it is found that the congenital and acquired cases were affected with about the same average frequency, when we consider the relative proportion of congenital to acquired deafness in our material; that is, as 18 congenital is to 33 acquired. In

form III. (complete deafness with positive irritability of the static labyrinth) the congenitally-deaf outnumber the acquired.

This corroborates the results obtained by Alexander and Kreidl, from which we may conclude that form III. represents a type of anatomical changes found in animals with congenital labyrinth anomalies (degenerative atrophy of the pars inferior labyrinthi).¹

It is interesting to note that in form IV. we find the vast majority to be cases of acquired deafness. This group further illustrates the circumscribed labyrinth affections; that is, complete non-irritability of the static labyrinth with rests of functions in the acoustic labyrinth, a well recognized *post-embryonal* process. This fact, contrary to the opinion of Herzog,² deserves to be emphasized.

Galvanic reaction: We found 21 cases with negative reactivity of the static labyrinth showing positive equilibrium disturbance. Most of these required a current of from 8-10 milliamperes to produce a positive reaction, while the remaining 30 cases with more intact static labyrinths showed typical reactions with a current of from 4-8 milliamperes, corresponding in its intensity of reaction to that obtained by turning upon the turning stool.

Equilibrium disturbance was pronounced in all 15 cases with complete non-reactibility of the static and acoustic labyrinth. This was also pronounced in the 7 cases with negative reactivity of the static labyrinth with hearing rests (group IV.). Somewhat less pronounced equilibrium disturbances were found in 10 other cases where the static labyrinth showed positive though diminished reactions to turning and to the galvanic current. Little or no disturbance of equilibrium was found in the remaining 21 cases, in which the reactivity of the static

¹ This fact has also been verified by Hammerschlag (*Z. f. O.*, Bd. 50).

² *Labyrinththeilung und Gehör*, München, 1908.

labyrinth to turning and the galvanic current proved normal.

Regarding equilibrium disturbances, the Alexander modified goniometer proved a more accurate apparatus for the detection of equilibrium disturbances than any of the other methods. It goes without saying that the figures contained in the large table¹ represent the average obtained and in almost every case verified by repeated examinations. In cases of labyrinthine disease and also frequently in normal people, there occur, with inclination of the goniometer, swaying movements, in spite of which, however, the patient is able to retain equilibrium. In these cases the swaying of the patient is mostly temporary and disappears with higher elevation of the goniometer. This condition was also found in a number of our deaf-mutes with remaining reactivity of the static labyrinth.

With a considerable number of pupils this swaying was entirely absent; the examined pupils, with less degree of elevation and without previous swaying, apparently lost their equilibrium completely, and threatened to fall. With these pupils a pronounced reaction appeared also when the inclined goniometer was lowered to the horizontal plane, whereby the pupil again threatened to fall. *A further fact worth noting, is that none of these pupils complained of any form of vertigo; accordingly, we cannot believe the equilibrium disturbances in these cases were dependent upon the semicircular canals, and yet we have here well recognized equilibrium disturbances of vestibular origin² (macula utriculi, macula sacculi). Furthermore, all of these cases showed absolute non-reactivity of the static labyrinth.*

Vision was more or less impaired in 7 cases (Nos. 5, 20, 22, 23, 28, 37, and 42). See Table II.

¹ S. Monatschr. f. Ohrenheilk., 1908.

² See G. W. Mackenzie, Arch. f. Ohrenheilk., 1908.

TABLE II.

Case No.	Eye Findings.	Equilibrium.	Reactibility of the Static Labyrinth.
5	maculae corneaë	disturbed	negative.
20	vision diminished	disturbed	left side negative, right side positive.
22	uncorrected high myopia	undisturbed	positive.
23	left-sided abducens paralysis	disturbed	negative.
28	double-sided corneal opacities after keratitis interstit. (lues hereditaria)	disturbed	negative.
37	maculae corneaë et strabismus convergens oc. dex.	slightly disturbed	positive.
42	atrophic bulbus O.D.	undisturbed	positive.

According to the above table, of 7 cases with impaired vision, we find: four cases with pronounced disturbances of equilibrium, one with slight disturbance of equilibrium, and two with normal equilibrium. The labyrinthine character of these equilibrium disturbances (excepting Case 37) and their independence of vision are recognized when one considers, in the above table, the reactibility of the static labyrinth. In the Cases 22 and 42 no equilibrium disturbances were present. Both of these showed positive reactibility of the static labyrinth. In Cases 5, 20, 23, and 28, the disturbances of equilibrium corresponded to the non-reactibility of the static labyrinth. In one case only (No. 37) was slight equilibrium disturbance found with positive reactibility of the static labyrinth. In this case we must conclude that the strabismus and macula corneaë were etiologically contributing factors of the equilibrium disturbance. At all events this case proves, in our examinations for labyrinthine equilibrium disturbances, the necessity of including a careful examination of the eyes.

Pupillary reaction: Pupillary reaction was absent in case No. 42 because of blindness and anterior synechia in

an atrophic eyeball. In one case (Slavik) the pupillary reaction was diminished upon one side (keratitis interstitialis from lues hereditaria). In the remaining cases the pupils were round and the reactions were prompt and normal.

Co-ordination: In five cases marked awkwardness or clumsiness was shown; however, without demonstrable ataxia. These five cases were all cases of acquired deafness, of which two (Nos. 20 and 44) were deafness after meningitis; one case (No. 38) after convulsions (probably meningitis); one case (No. 42) after measles; one case (No. 46) after typhoid fever. Considering that in the remaining 11 cases of deafness after meningitis there existed no clumsiness, we can see no reason why the clumsiness in cases Nos. 20 and 24, as well as in the remaining cases, is in any way characteristic for meningitis or the cortical changes following meningitis. In Case 42 there existed in spite of the clumsiness no equilibrium disturbances, in the remaining four cases equilibrium disturbances were present; however, not more marked than in the great number of cases of equilibrium disturbances *without* clumsiness. It may be said that in the four cases (Nos. 20, 44, 38, and 46) the clumsiness contributed to, but in no way was the cause of, the equilibrium disturbances.

SUMMARY.

It is shown, from our material, that the static labyrinth is somewhat more resistant to deleterious influences than the acoustic labyrinth (cochlea); however, the difference is not so great as had been expected from previous anatomical examinations, especially in cases of malformation. The remarkable findings of degenerative atrophy of the pars inferior labyrinthi (most characteristic anatomical findings in congenital deafness) are herewith explained, that the pars superior, phylogenetically a much older part, is relatively more resistant to intra-embryonal-appearing pathologic changes (arrest of

formation) than the philogenetically young cochlea. That this applies in cases of congenital deafness had also been demonstrated in the material examined by us, and the findings, complete deafness with remaining irritability of the static labyrinth (form III., see page 508), show that of the nine cases seven were cases of congenital deafness.

On the other hand, the results of our examination show that there is no essential difference between the static and acoustic labyrinth in their powers of resistance to *post-embryonal* pathologic influences. In these 51 cases before us, isolated destructive processes occurred but slightly more frequent in the acoustic apparatus (cochlea, nerve, and ganglion spirale) than in the static labyrinth (sacculus, utriculus, semicircular canals, vestibular nerve, and its ganglion). It may be observed, however, that these figures possibly do not correspond exactly with the results that might be obtained from an examination of the total number of pupils in the deaf-mute institute. On the other hand, cases of isolated lesions of the static labyrinth with more or less reacting acoustic labyrinth—that is, with good hearing—are not to be found in deaf-mute institutes. These latter are to be observed rather in the clinics, where they come to seek relief from their vertigo and equilibrium disturbances.

If we summarize all cases with inner-ear symptoms (deafness, vertigo, and equilibrium disturbances), we find a slight majority of affections of the acoustic labyrinth over those of the static labyrinth. Since the relationship of turning vertigo (*Drehschwindel*) to the organ of hearing has been recognized, these patients seek the ear clinics more frequently than formerly. A very instructive case of this kind, which we also had opportunity of examining, is a case recently reported by Neumann: a patient, previously in good health, was taken suddenly with a very heavy attack of (*Drehschwindel*) vertigo and nystagmus to the sound side. The examination revealed, further, evidences of neuritis of the tri-

geminus with herpes zoster; so that we must accept the case to be one of isolated neuritis of the nervus vestibularis, since the nervus cochlearis was intact and hearing normal. Similar cases have been reported by Frankl-Hochwart and still another case by Neumann.¹

In many of the cases of complete destruction of the acoustic and static labyrinth or of the nervus octavus the equilibrium disturbances were very pronounced, all of which showed more or less positive Romberg (the most uncertain of all tests for disturbances of equilibrium); while, on the contrary, none of them had vertigo or gave the history of vertigo. The non-reactibility of the semi-circular canals, arrived at by rapid and long-continued turning (10 to 20 times) upon the turning stool, failed to produce, no matter in what position the head was held, either nystagmus or vertigo. We desire to lay special stress upon this finding, since it demonstrates that the late labyrinthine equilibrium disturbance is not, as has been generally accepted, an accompanying or resulting phenomenon of vertigo and nystagmus.

The methods adopted for the examination of equilibrium disturbances in these cases consisted in the performance of straight-line movements in the three directions of space (walking, running, hopping), and the examination upon the goniometer, all of which were made with both open and closed eyes. In all of these methods of examination (straight-line movements), irritation or examination of the semicircular canals is excluded.

We are enabled here to express the opinion that in the above cases the equilibrium disturbances are due to the failure of impulses having reached (which normally are carried to) the end organ in the vestibular apparatus (macula sacculi and macula utriculi). This opinion, unproven up to the time of our examinations, is supported by the fact that in our series of clinically and anatomically

¹ To be sure, also cases of isolated neuritis of the nervus cochlearis have been observed (Kaufmann and Hammerschlag).

(Alexander) examined cases, the degree of equilibrium disturbances was in direct proportion to the amount of destructive processes in the vestibule.

This leads us to a clinical confirmation of Breuer's theory: that the sacculus and utriculus together represent the nerve end organ for perception of motion acceleration in straight lines.

The impulses proceeding from the two nerve endings serve for the maintenance of equilibrium of the body and head in the three principal directions of space. This opinion has received, from a clinical standpoint, substantial support by clinical examinations made by G. W. Mackenzie. When the vestibular apparatus is destroyed, there occurs disturbance of equilibrium, which manifests itself upon active and passive movements in the three directions of space. An exact answer to the question of the function of the nerve endings in the vestibular sacs is possible only by experimental means. Up to the present time, however, we have not succeeded in producing, in mammals, isolated experimental changes in the vestibular sacs, preventing, at the same time, changes in the semicircular canals.

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ON THE STRUCTURE AND FUNCTION OF THE EPITHELIUM IN THE SULCUS SPIRALIS EXTERNUS.

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(With eight figures on Appended Plates A, B, C, D, and E.)

INTRODUCTION

THE outer wall of the ductus cochlearis is divided by the prominentia spiralis into two unequal concave segments. The larger segment lies above the prominentia spiralis and is occupied by the stria vascularis. The smaller segment lies below the prominentia spiralis and is known as the sulcus spiralis externus.

The epithelium covering the outer end of the membrana basilaris is composed of tall cylindrical cells called the cells of Claudius. These cells extend beyond the attachment of the basilar membrane, over on the crista of the ligamentum spirale, and in this way cover in part the sulcus externus.

The epithelium covering the prominentia spiralis is made up of a single layer of low cuboidal cells. These cells are continuous above with the epithelium of the stria vascularis. They extend downward well into the sulcus externus, and in the labyrinth of the pig consist, as a rule, of from six to eight rows.

Continuous with the epithelium covering the prominentia spiralis and covered over in part at times by the cells of Claudius is a group of epithelial cells which occupy

the deepest part of the sulcus externus. They possess certain marked peculiarities which distinguish them from epithelium found elsewhere in the labyrinth. These cells have been observed by Deiters and studied by Boettcher, Gottstein, and Katz, and later by Prenant and Retzius. The peculiar feature of this epithelium as described by these writers was the presence of long finger-like processes from the cells, extending out into the connective tissue of the ligamentum spirale.

As regards the function of these cells, Gottstein was at first inclined to look on them as a type of neuro-epithelium, but was unable to establish any connection with nerve fibres.

Boettcher believed they had contractile properties, and attributed to them the important function of accommodation in the ear by putting the basilar membrane on tension.

Katz was inclined to agree with Boettcher in the contractile nature of these cells.

Prenant accepted the theory that these cells represented muscular tissue derived from epithelium.

Retzius did not believe that any evidence had been produced that would justify the conclusion that these cells are contractile.

The latter investigator succeeded in staining these cells by the Golgi method in the mouse five to eight days old and in the foetus of the cat. He gives the following careful description of their structure.¹

Diese letzteren Zellen dringen tief in das unterliegende Gewebe hinein; die Endäste dieser Fortsätze dringen hier und da fast durch die bindegewebige Wand bis zur Nähe ihrer Aussenfläche. . . . Die Zellen, deren oberes Ende in der oberen Fläche des Epithels steht und dem oberen Ende gewöhnliches Cylinderzellen entspricht, radiiren von hier aus nach aus-

¹ "Über das Epithel des Sulcus Spiralis Cochleae." *Biologische Untersuchungen*, Neue Folge v., 1893.

senunten, nach aussen und nach aussenoben; Fortsätze, die sich in verschiedener Weise verzweigen und sich hier und da kreuzen, dringen tief in das Bindegewebe hinein. Ein Anastomosiren der Zellen-Fortsätze unter sich, das Prenant also möglich erwähnt, sah ich nicht; noch weniger habe ich von dem von den Autoren angenommenen Zusammenhang dieser Zellen, resp. Zellen Fortsätze mit den umgebenden Bindegewebszellen jemals eine Spur angetroffen.

Der Kern dieser eigenthümlichen Zellen liegt in der Regel unter der unteren Grenze der gewöhnlichen Epithelzellen des unteren Sulcustheils; man findet ihn gewöhnlich in einer spindelförmigen Erweiterung des Zellen-Körpers, die tief in dem Bindegewebe liegt. (P. 39.)

AUTHOR'S INVESTIGATION.

My attention was first directed to the peculiarities of the epithelium in the sulcus externus while examining sections of the labyrinth of the adult guinea-pig which had been stained by the Mallory reticulum stain. It was often noted that in sections stained by this method a group of epithelial cells occupying the bottom of the sulcus externus would be sharply differentiated from the neighboring cells. The cells thus differentiated not only sent long finger-like processes into the spiral ligament, but there was an actual invasion by the cells themselves of this structure. The picture presented by these sections was such a striking one (see Fig. 1) that I was led to undertake a closer investigation regarding the development, the exact character, and especially the functional significance of these cells.

The material used in this work was for the most part the labyrinth of the domestic pig.

In the ear of the pig these cells first began to differentiate from the remainder of the epithelium in this region in the embryo measuring about 12cm long. At this age protoplasmic processes from the single layer of cells lining the sulcus began to perforate, in the basal coil, the strong basement membrane separating these cells

from the spiral ligament. In the pig embryo 15cm long, such processes had formed from these cells throughout the coils of the cochlea and in the basal coil the bunching up of the cells began to appear preliminary to the actual invasion by the cells themselves of the spiral ligament.

In studying the labyrinth of the new-born pig and the foetus at full term, which may be looked upon as representing the adult condition, the following characteristics of this epithelium were noted.

In the first place, the cells in the sulcus externus, as regards staining properties both of the cell protoplasm and the nuclei, resemble closely the epithelium covering the prominentia spiralis and are sharply differentiated from the cells of Claudius, which latter take, on the whole, a much fainter stain.

In the second place, the epithelial cells of the sulcus externus as they penetrate the spiral ligament are always the more clearly differentiated from the surrounding structure the closer they lie to the free surface of the sulcus. This demarcation is often accentuated by a slight cleavage between these cells and the connective tissue of the spiral ligament, due to a slight shrinking in the process of preparation. On the other hand, the deeper these cells penetrate into the spiral ligament the more difficult it becomes often to distinguish them from the neighboring structure.

The depth to which the nuclei of these cells penetrate the spiral ligament often extends to fully midway between the sulcus externus and bony capsule. The fibrillar processes which are apparently continuous with the deeper ends of these cells extend usually to the loose reticulum of cells bordering the bony capsule. The processes usually run outward and upward. A bundle of these processes, shown in Fig. 3, ran parallel to the stria vascularis up to the attachment of the membrane of Reissner.

In the third place, it was observed that these epithelia

cells which invade the spiral ligament do not form a continuous band around the coils of the cochlea, but appear in clumps. Between these clumps the epithelium lining the sulcus externus presents a single layer of cuboidal cells quite like the epithelium covering the *prominentia spiralis*. It was also noted that a depression was usually found on the free surface of the sulcus externus, marking the point at which such a clump of epithelial cells penetrates the spiral ligament. These characteristics are especially clearly shown in sections cut parallel to the long axis of the coil. See Fig. 4.

Finally, I observed that often where sections were made so as to cut across an epithelial clump at right angles to its long axis, a small clearly-defined central lumen could be made out. This tubule was usually so small that sections cut parallel to the long axis of the epithelial clump usually missed it completely or else cut it obliquely so that its presence was easily overlooked. Examples of sections cut in this way, showing the epithelial clumps but no tubules, are shown in Figs. 1 and 2. It is evident that sections such as these must be the usual ones, and it is this formation that has been observed and described by previous writers.

After the presence of tubules in these epithelial clumps became known to me, I had no difficulty in finding a large number of sections in which they could be made out. To find sections, however, in which such tubules could be traced to the free surface of the sulcus externus was no easy task, since to cut the tubule parallel to its long axis was largely a matter of chance. Among the many hundred sections I have cut and stained since I first reported the finding of these tubules,¹ I have a few which show the tubule throughout a large part of its course. Several of these I have had drawn, see Figs. 3, 6, 8.

This tubule is sometimes found dilated. In Fig. 5

¹ "Proceedings of the Association of American Anatomists," *American Journal of Anatomy*, vol. v., No. 2, p. vii., 1905.

is shown a preparation where this dilated tubule has been cut across in such a way that the cement lines (Schliessleisten) marking the cell-boundaries on the free surface of the tubule are shown. It sometimes happened that the tubule underwent a marked cyst-like enlargement, just as one might expect in case secretion was retained by an occlusion of the tubule. Such a cystic enlargement is well shown in Fig. 7.

As regards the function of these peculiar epithelial clumps in the sulcus externus, I was convinced early in my study of the cells that the theory that this was a contractile structure for the purpose of exerting tension on the membrana basilaris was untenable. The reason was that, while these cell-processes were found scattered along the entire length of the ductus cochlearis from the beginning of the basal coil to the upper coil near the apex of the cochlea, it was for only a very short area in the basal coil that the position and direction of these cell-processes were such that their contraction could possibly exert tension on the basilar membrane.

That the cells have an important physiological function there can be no doubt. In my earlier studies of the blood-supply of the labyrinth,¹ I had been impressed with the elaborate system of blood-vessels for supplying the ligamentum spirale, and especially with the wonderfully rich capillary supply to just this part of the ligament into which these cells penetrate. It seemed improbable that a structure having a purely passive supporting function, such as is usually attributed to the spiral ligament, should require the rich capillary supply which had here been provided. At that time I attempted in vain to surmise what the significance of this rich blood-supply was, what important function resided in this part of the ligamentum spirale which called for this

¹"The Distribution of Blood-vessels in the Labyrinth of the Ear of *Sus Scrofa Domestica*." *The Decennial Publications*, University of Chicago, University of Chicago Press, 1903.

extensive capillary supply. The suggestion has sometimes been made that the varying degree of tension of the radiating fibres of the membrana basilaris, which seems to be necessary if we accept the teaching of Helmholtz that these fibres act as resonator, was dependent on the filling of these blood-vessels in the spiral ligament. It does not seem probable, however, that nature would be forced to resort to this indirect method of accomplishing an end so easily reached in other ways. I might add, furthermore, that in a recent study of the membrana basilaris I believe I have been able to demonstrate that this structure cannot act as a vibrating mechanism, but that it is merely a passive supporting apparatus.¹

With the discovery that these epithelial clumps growing out from the sulcus externus into the spiral ligament are provided with tubules which open on the free surface, the question of the function of these cells becomes quite clear. Such tubules can have but one significance, that of a secreting mechanism. We have here in the sulcus externus epithelial prolongations each provided with a central lumen—in other words, a type of secreting or glandular epithelium. Whether these glands secrete all of the constituents of the endolymph or only a part, we have no means of knowing. It seems probable, however, that the stria vascularis may share in the function of secreting the endolymph.

EXPLANATION OF PLATES.

Terms Common to All the Figures.

- S s e = Sulcus spiralis externus.
- P s = Prominentia spiralis.
- C C = Cells of Claudius.
- S v = Stria vascularis.
- L s = Ligamentum spirale.

The figures are all drawn with the assistance of the Leitz Obj.
 $\frac{1}{12}$ Immers.

¹"A Restudy of the Minute Anatomy of Structures in the Cochlea, with Conclusions Bearing on the Solution of the Problem of Tone Perception." *The American Journal of Anatomy*, vol. vii., No. 2, pp. 245-257, Aug., 1907.

FIG. 1.

Preparation from the labyrinth of an adult guinea-pig, fixed in Zenker's solution, embedded in paraffin, and cut five micra thick. The section has been stained with the Mallory reticulum stain. This preparation shows a clearly defined clump of epithelium from the sulcus externus penetrating deeply the spiral ligament and sending out long finger-like processes which extend to the loose reticulum of cells bordering the bony capsule.

FIG. 2.

Preparation from the labyrinth of the new-born domestic pig, showing the epithelium of the sulcus externus growing out into the spiral ligament. A slight cleavage separates these epithelial cells from the surrounding structure.

FIG. 3.

Section of the outer wall of the ductus cochlearis from the labyrinth of a new-born domestic pig. Several clumps of epithelium from the sulcus externus have grown out into the spiral ligament. The lower clump is cut so as to show the central lumen throughout a large part of its course. The upper clump is not cut so as to show its tubule. Long fibrillar processes extend from this clump of cells parallel with the stria vascularis up as far as to the attachment of the membrane of Reissner.

FIG. 4.

Section cut parallel to the long axis of the basal coil of the cochlea and passing directly through the sulcus externus. Successive clumps of epithelium growing out from the sulcus externus into the spiral ligament are shown. In several of these the central lumen has been cut across. The preparation is from the foetus of a domestic pig 18cm long.

FIG. 5.

Preparation from the labyrinth of a new-born domestic pig, fixed in Zenker's solution, embedded in celloidin, cut 10 micra thick, and stained with neutro-gentian. This section is cut through the centre of a dilated tubule which occupies the middle of a clump of epithelium growing out from the sulcus externus into the spiral ligament. The cement lines (Schliessleisten) marking the cell boundaries in the free surface of the tubule are clearly defined.

FIG. 6.

Section from the labyrinth of a new-born domestic pig, cut close to the beginning of the basal coil. A tubule in a clump of epithelium from the sulcus externus is shown throughout a large part of its extent and opening on the free surface.

FIG. 7.

Section from the labyrinth of a new-born domestic pig, showing a cystic dilatation of the tubule in a clump of epithelium from the sulcus externus. The cell boundaries on the free surface of the tubule are shown.

FIG. 8.

Preparation from the labyrinth of a new-born domestic pig. Section shows an extensive invasion of the spiral ligament by the epithelium occupying the sulcus externus. The section chanced to cut through the central lumen of an epithelial clump throughout a large part of its course.

ARTERIAL HEMORRHAGE FROM THE AUDITORY
MEATUS IN A CHILD, AFTER A BRIEF AT-
TACK OF OTITIS MEDIA ACUTA FOLLOWING
TONSILLITIS WITH TONSILLAR ABSCESS.
LIGATION OF THE COMMON CAROTID: UN-
EVENTFUL RECOVERY.

BY DR. J. A. SPALDING, PORTLAND, ME.

CASES of abundant and alarming arterial hemorrhage from the auditory meatus, calling for ligation of the common carotid, in chronic otitis suppurativa are so rare that each one should be carefully reported. Even more urgent is this need when the operation is called for under similar hemorrhages following cases of otitis acuta in children. The case which I now report comes under the latter category, and deserves brief mention as an addition to the small list of similar cases hitherto recorded.

During the service of Dr. W. B. Moulton at the Maine General Hospital in 1907, a patient was brought in with the following history.

A boy of five years of age three weeks before entering the hospital had suffered from an attack of tonsillitis followed rapidly with a tonsillary abscess on the right side of the throat. After this had spontaneously discharged a large amount of pus, otitis acuta set in on the same side. The boy had never before had any discharge from either ear and his hearing in both ears, so far as the parents had observed, had always been perfect. After a day of considerable pain in the ear the *Mt* perforated and gave issue to a large amount of steadily-flowing serous discharge, but not tinged with

blood. This continued until four days before the child was brought to the hospital. Here then we have about sixteen days in all, for the tonsillitis, the abscess, the otitis, and the serous discharge.

On the seventeenth day without previous warning there was a sharp arterial hemorrhage from the right meatus, and so abundant that a physician was sent for. He plugged the meatus with the result that blood flowed from the nose and mouth whilst considerable ran off into the stomach. The bleeding ceased when the child fainted from loss of blood. On three successive days similar hemorrhages occurred with abundant loss of blood. On the fourth day the child was admitted into the hospital.

Condition.—Very pale, delicate in appearance. Hæmoglobin 40. Extreme pallor of the entire body. Considerable swelling over the tip of the mastoid and extending down into the neck below the ear.

Consultation being at once held it was decided to postpone ligation of the common carotid owing to the dangers implied by the operation, and to try farther plugging of the meatus with adrenalin and to inject chloride of calcium hypodermatically. This was faithfully carried out, but a fresh and abundant arterial hemorrhage from the meatus the next morning made ligation of the common carotid seem imperative at every risk.

Before the operation was performed by Dr. John F. Thompson, then on surgical duty at the hospital, the following points in the child's condition were noted. Right pupil contracted to pin-head size; left pupil greatly dilated. Patient apparently comatose from loss of blood. Temperature 102°. Pulse difficult to count but thought to be about 170. Respiration very slow and hardly perceptible. After etherizing, the common carotid was tied in the usual manner and the mastoid opened and carefully cleansed of some small amount of pus and considerable debris, by Dr. Moulton.

The patient improved the moment that the operation was finished; the pupils became of equal size, the pulse rapidly normal, and temperature also fell to normal. The child made an absolutely uneventful recovery; the mastoid dressings were changed once or twice and in a fortnight the

patient passed from under observation. There has been no recurrence since.

Owing to the extreme rarity of such cases a few remarks on the literature may not come amiss.

Milligan (*Practitioner's Handbook of Diseases of the Ear*, sixth edition, p. 350) says that ulceration in the bone in otitis 'suppurativa chronica may extend into the carotid. Almost instantaneous death has been recorded, but generally three or four abundant hemorrhages precede the fatal one. The common carotid has been ligated, but owing to recurrence of hemorrhage ligation of both common carotids has been done. If the hemorrhage diminishes by carotid pressure the vessel must be ligated, but if it does not diminish then the bleeding is venous and may be arrested by plugging.

Politzer (*Handbook of Diseases of the Ear*, Am. Ed., 1901, p. 504) says this accident is of the greatest rarity and mentions instances from the practice of Kessler and Sutphen. His own personal case was in a tuberculous man and the autopsy showed erosion of the carotid in the carotid canal where the vessel curves from vertical to horizontal position. Baizeau and Choyau's cases are also mentioned, as well as an instance of such hemorrhage with an intact *Mt*, the hemorrhage flowing into the pharynx. Tuberculosis, syphilis, and scrofula are suggested as causes of the carotid erosion and in one instance the bleeding followed a chemical injury. The bleeding may last for a few minutes or oozing may continue for several hours. A patient who refused to permit ligation died after recurrent hemorrhage in eleven days. Politzer mentions Billroth's case which will be mentioned below, and suggests that in hospital cases a nurse should be on hand to apply compression to the carotid, and to try the effect of hemostatics, on which, however, he places little reliance.

The most careful study of hemorrhages from the

auditory meatus has been made by Spencer (*Med. Chi. Trans.*, 1901, lxxiv., p. 373), who goes over the whole ground so far reported and adds a new case of his own. His paper, greatly condensed, shows that there are three classes of patients in whom hemorrhage has been observed: the tuberculous with long-standing otorrhœa, the healthy with otitis suppurativa chronica, and children with otitis acuta. His list includes twenty cases and to these he adds one. The present case would make the number *twenty-two* so far recorded in accessible literature.

His earliest case is from Toynbee. He also quotes from Schwartze and Politzer. Death in one instance resulted from a furious hemorrhage, a sequestrum putting the carotid into free communication with the large cavity. A tuberculous patient had long suffered with suppurative otitis and the discharge had often been tinged with blood. Another patient had a violent fit of tuberculous coughing which was followed by hemorrhage ultimately fatal. All of the tuberculous patients died, some despite ligation of the carotid.

Spencer's *second class* contains those occurring in otitis suppurativa chronica, concerning which he says that owing to better treatment of the cause now prevailing, hemorrhages are likely to become more rare. Most of these seem to have been fatal; though ligation was not employed in any except in one of Billroth's in which ligation of both common carotids was performed because ligation of one alone did not check recurrence of the hemorrhage. His *third class* contains a list of six cases of otitis acuta in two of which death followed hemorrhages occurring very rapidly after the onset of otitis acuta but in which ligation was not done at all. Finally he adds four cases in which ligation was done and in all of which recovery took place. The youngest child was three years old, the oldest eleven. The time between the onset of the otitis and the hemorrhage varied from ten days to two months.

The discussion following Spencer's paper showed the rarity of the occurrence and defended the propriety of ligating the common carotid despite the dangers of the operation advanced by some members. There was also a general consensus of opinion that, if the patient recovered, the less any attempt was made to treat the local conditions of the ear, afterwards, the less the chance of instituting meddlesome surgery. Ligation of the internal carotid was regarded as superfluous; besides the operation on the common carotid lies farther away from any source of infection, and the operation is easier. Regarding the risks of ligating the artery in children, Spencer says that after studying a long list of such operations on children for other causes than the hemorrhage which he has just described, he could not find any great percentage of mortality. The operation of ligating the common carotid is, however, not to be undertaken lightly, because an examination of statistics for all diseases in adults and in children shows an average mortality, when done for wounds, of 54%; when done for exophthalmos, of 7%; and for all possible varieties of disease and injuries the recorded mortality as I find between 1900 and 1906 was 21%.

These remarks contain notices, in brief, of all the instances of hemorrhage from the auditory meatus which I have, personally, been able to discover in my accessible literature. It would be well worth while if some of our younger students in otology in the larger centres were to investigate this topic, since the appearance of Spencer's paper in 1901.

TWO CASES OF SINUS THROMBOSIS, WITH ATYPICAL SYMPTOMS. OPERATIVE AND PATHOLOGICAL FINDINGS.¹

BY S. J. KOPETZKY, M.D., NEW YORK CITY.

CASE I.—C. W., aged 43, appeared at the Manhattan Eye and Ear Hospital on October 8, 1907, giving a history of having a discharging ear, which had persisted for one year, having been caused by a severe cold. The examination shows a perforation of large size, marginally situated, through which a moderate amount of purulent discharge came away. The patient was put upon rational cleansing treatment and seemed to improve as time went on. On March 20th, he appeared at the Clinic with a swelling over the mastoid process of the left side, stating that this swelling had appeared two days previously. He had no pain and no fever and had passed a comfortable night. The swelling extended well over the temporal and parietal regions and forward as far as the outer angle of the eye. The external auditory canal showed oedematous infiltrate.

Believing that I was dealing with an acute exacerbation of chronic otitis media, I suggested operation and immediately sent him to the ward for preparation, and within a half hour the operation was performed.

Status on Admission: Temperature 100.2°, R. 20, P. 80.

Operative Findings.—The usual post-auricular incision demonstrated no pus from the swollen oedematous tissue over the mastoid process. This was the first intimation that I had that I might be dealing with some sinus trouble, as previous observation on sinus cases had shown this finding

¹ Read before the Section on Otology, N. Y. Academy of Medicine, October 9, 1908.

in sinus involvement. (*Annals of Otology*, March, 1908.) The removal of the cortex showed the mastoid apophysis filled with fluid pus. The cortex overlying the antrum was rather thick. The mastoid cells extended backward and upward rather unusually far and were found filled with necrotic debris and pus. The sinus took a course rather forward and almost bisected in a diagonal direction the mastoid process, cells being evident both above and below it. The sinus wall was covered with necrotic granulations and its exposure for considerable distance failed to show a healthy wall. The sinus was then opened at the level of the antrum and found to contain a dark firm clot. The sinus was now traced backwards toward the torcular for about an inch and a half, being opened all along its route, and free bleeding was obtained about $1\frac{1}{2}$ inches back from the knee. Working downward toward the bulb the central end of the clot was not reachable, therefore after temporarily packing the cavity, resterilizing our hands and instruments, the neck was prepared for resection of the jugular.

The operation on the jugular was easily performed, the internal jugular resected from just above the clavicle to above the facial vein. The clot was now removed from the bulbar end of the sigmoid sinus and free bleeding followed probably from the petrosal. Both wounds were cleaned, mastoid wound packed in the usual manner, and the lips of the neck wound approximated and ligated with one surgical knot, leaving *in situ* a strip of iodoform gauze to act as a drain. The neck wound rapidly healed; the mastoid wound took the usual course, and healed; the middle ear became dry without incident. Patient eventually discharged cured.

The temperature variation of the first week was atypical, the temperature at no time going above 100° ; R. and P. in proportion.

This case is presented to demonstrate how extensive an involvement of the sinus may be present without giving the usual up-and-down temperature or any other signs to point to the gravity of the patient's condition. Here is a man who walks into the clinic with a clot in his lateral sinus at least three to three and a half inches in

length. Secondly, the only physical sign was an oedematous condition over the mastoid region and extending well beyond it.

To point the lesson of this case more thoroughly, I present Case 2.

CASE 2.—B. S., aged six, came under observation June 10, 1908, at the Ear Clinic of the N. Y. Nose, Throat, and Lung Hospital, giving a history of having had scarlet fever two years previously, from which he recovered with a persistent purulent otorrhoea from his right ear. At the time of his appearance at the Clinic the right ear was profusely discharging a foul-smelling thick creamy pus. The left ear was examined and found normal. Cleansing antiseptic treatment was instituted, and this was continued until June 15th, when the mother reported that he had vomited during the previous day, had passed a restless night, had had some temperature, and complained of pain in the right ear. During the examination he vomited excessively and seemed a very sick child. T. 101.6°, P. 120, R. 26.

I sent him to the Red Cross Hospital for observation; because the local condition in his ear, the absence of any mastoid tenderness on either side, and the free discharge of pus, did not seem to me to be the cause of his trouble. On the other hand, a thorough physical examination by the attending physician of the hospital, Dr. L. K. Neff, gave negative results. He was kept under observation until June 18th, when, because of a beginning swelling of the external auditory canal of the right ear, the radical operation was performed; his condition being summed up as an acute exacerbation of his chronic otitis media on the right side.

Operative Findings.—The retraction of the first part after the usual mastoid incision showed a well developed mastoid outline, cortical landmarks well defined. The mastoid process was found partly eburnated, but there were well marked cells radiating about the tympanic cavity and mastoid antrum. These cells were filled with fluid pus and with pseudo-cholesteatomatous masses. An exposure of the sigmoid portion of the lateral sinus was effected at its knee for about $\frac{1}{4}$ inch square. The wall was found to be healthy.

The usual technical procedure to take the post-auricular wall, eviscerate abnormal mastoid and tympanic contents, and cut a Panse flap with primary suture of the post-auricular wound then followed.

It is to be noted in passing that the mastoid process presented no deviations from the normal process of a child six years of age. The day following the operation we find the temperature normal, R. 74, respiration 22. Removal of the outside dressing showed the wound to look healthy. On the night of the second day, the temperature was still normal, but the pulse rose to 102°, R. 26. The child was slightly restless during his sleep, although nothing of an unusual character was observed; occasionally the patient complained of pain in the ear which had been operated. On the morning of the third day after operation the patient was sitting up in bed, crying that he was hungry, presenting normal T., P. 104°, R. 24. At 8.30 that day he had vomited after having taken some gruel. Towards evening he became very restless, had a crying spell, and in a seeming frenzy had torn off his bandages, nevertheless at 11.30 that night he was sleeping, and the evening condition showed T. 101.2°, P. 122, R. 20. The nurse reported a slight attack of what she termed delirium earlier in the evening. On the morning of the 4th day, I found the patient lying on his back perfectly relaxed and unconscious. T. 102°, P. 132, R. 22. His condition alternated between periods of unconsciousness and periods of crying. There was no Koenig sign, no opisthotonus.

The dressings were removed, the stitches taken from the wound, and the entire cavity inspected. The wound was found clean with no signs of pus.

Thinking that there might be some local condition in the meninges or in the sinus (although there were no symptoms to justify such a diagnosis), a few whiffs of chloroform were administered and the tegument removed exposing the meninges, and the bony sinus wall was also removed to expose the sinus. Both were found healthy in appearance and further efforts were temporarily stopped. The child died that afternoon. Tentatively a diagnosis of rupture of a brain abscess was made. Permission for an autopsy was obtained and this showed the meninges normal, no increase in cerebral fluid;

sections of the brain failed to reveal any brain abscess and showed nothing characteristic although many small punctate hemorrhages were noticeable throughout its substance. The wound in the mastoid showed that it had been completely exenterated and an examination of the labyrinth was negative. The sinus on that side was found normal. In the removal of the brain the meninges of the opposite temporal bone were left *in situ*, and as a last resort, while looking for the cause of death, this was pulled from the cerebral surface of the temporal pyramid. In doing this, the left sinus was uncovered and torn, and we were surprised to find within it a yellow semi-liquid mass. The skin over the left mastoid was then retracted, cortex exposed, and realizing then that I was dealing with a thrombosis of the lateral sinus on the left side of the head I endeavored to open the left mastoid. The first stroke of the chisel showed the sinus covered only by a thin cortex, and further efforts were stopped until we had extracted the temporal bone. How far toward the torcular the clot extended we could not make out, as part of the mass was lost in the debris following the removal of the brain. It completely filled the sinus from the knee to the bulb and beyond. The thrombus was partly organized and yellow, and from its macroscopical aspect was pronounced by the pathologist, Dr. Gonzales, a fatty degeneration of the thrombus. He estimated that it had been *in situ* for at least a week or ten days. The specimen I present is of this left ear. It showed the middle ear filled with pus and detritus, the antrum small, and no other cells evident at all. The sigmoid sinus lies directly under the cortex and takes up the entire partly rudimentary mastoid tip. There is no evidence of a real mastoid cell, if we leave out of account the small space directly under the mastoid fossa, and at the angle formed by the two sides of the petrosal pyramid. The contrast of this side to that upon which I operated is very marked, aside from the other peculiarities of this case.

Here then was a case in which a thrombus developed during an acute invasion of both ears, in which marked pyæmic symptoms were absent. On the right side, because of its previous history and local findings and

because of the pain, an operation was undertaken; the left side, because of congenital mal-development and resulting anatomical peculiarities, permitted pus to travel direct from the tympanic cavity to the sinus, which was in almost juxtaposition, and a thrombus was thus engendered. This thrombus was evidently not of a very infectious variety and became organized, and then, either because of the trauma to the head during the operation on the opposite side or because of some other extraneous factor, it underwent fatty degeneration, and from the dissemination of this fatty material the patient succumbed.

At no time during the patient's illness was there any complaint of pain in the left ear, and when the other ear was bandaged the patient seemed easily to hear ordinary conversational tones with his left ear up to the very time that he became unconscious. There seems to be a need for an analytical study of all the atypical cases of sinus thrombosis in order that some data of diagnostic value may be accumulated.

A CASE OF UNSUSPECTED NECROSIS OF THE
PETROUS BONE. MENINGITIS. DEATH.
CO-EXISTING MASTOID SUPPURATION AND
EXTRADURAL (CEREBELLAR) ABSCESS.

BY HILL HASTINGS, M.D., LOS ANGELES, CAL.

(With one photograph on Text-Plate XV.)

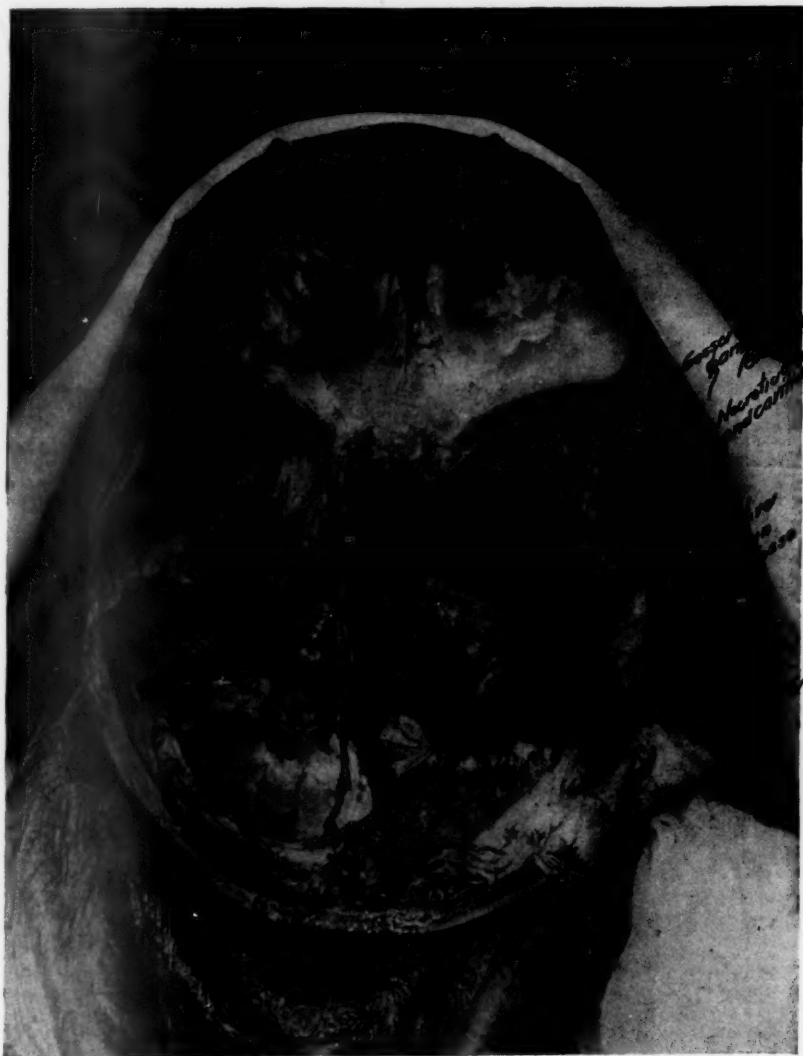
A REPORT of this case is made because of the failure to diagnose, before the mastoid operation, the suppuration of the internal ear which likely existed and which with necrosis of the petrous bone was found at autopsy. The perisinus abscess, with involvement of the cerebellar dura found at operation, was accepted as the cause of the fatal meningitis. A similar mistake likely occurs occasionally with most of us, and is only discovered by a routine examination of the petrous bone at autopsy.

T. K., age seventy-two, admitted to the Good Samaritan Hospital, June 20, 1908.

Complaint.—Ear discharge and "neuralgia of the head."

History.—Onset was four months ago. Abscess in the right ear followed a severe attack of grippe; suffered considerably. The ear discharge has been constant. The past week or two the discharge has been profuse, requiring irrigation every two hours, and running out on the pillow at night. Pain of a neuralgic character has been present since the beginning of the ear abscess, referred to the mastoid and temporal regions of the right side. Of late (the past two or three weeks) the pain has been referred also down the neck

ILLUSTRATING DR. HASTINGS'S ARTICLE ON "A CASE OF UNSUSPECTED
NECROSIS OF THE PETROUS BONE," ETC.



and into the right eye. No swelling over the mastoid has been noticed. No dizziness. Deafness has been profound (claims he has never heard well in the right ear since an abscess in this right ear twenty-four years ago, which healed up promptly). Prior to the present trouble there had been no ear discharge. General condition has been poor; delicate, feeble man, addicted to the use of liquor. Has not been confined to bed entirely. Has not felt as well as usual, but not sick. His temperature has not been taken or recorded and no special notes had been made inasmuch as his condition did not seem in any way alarming.

There has been no chill or high fever, but of late slight fever and some sweating at night have been remarked. There has been no stupor, drowsiness, or vomiting. One week ago had some nausea and vomiting which was attributed to his stomach; no appetite; constipated. No trouble with the bladder. No history of kidney trouble.

Examination.—Patient walked into the hospital and has not gone to bed. Feeble and looks his age. Pulse 110 with some arterial thickening. Temperature 99°. Tongue fairly clean. Mastoid: marked thickening around and behind the mastoid on the right side; tenderness marked on pressure over the mastoid and acute over the point of exit of the emissary vein. No thickening along the jugular. Meatus full of thick pus with a small perforation below and in front of the umbo through which pus continually oozes; pus in the canal had a slight streak of blood. His brother (a physician) states once or twice before there has been a little blood in the pus. No dizziness. Hearing negative to watch and doubtful to conversation. (Smears and a culture taken from the pus showed streptococci.) Examination of the eyes failed to show choking of either disk, but marked injection of the right disk with indistinct margin. Immediate operation advised. (Suspicion of intracranial involvement.)

June 21st.—Operation at Good Samaritan Hospital showed the existence of a perisinus mastoid abscess. On removing the cortex the mastoid found full of pus; welled out under considerable pressure; pulsating. Put throughout the mastoid, with large abscess down on the descending limb of the lateral sinus. The tip found entirely broken down into one

large abscess cavity extending through the inner table into the digastric fossa. The abscess extended from the sinus knee to the tip. Sinus exposed through necrotic softening of the bone and uncovered throughout its descending limb; its wall was thick, as was also the adjacent dura over the cerebellum; no thrombus believed to exist. Slight facial twitching occurred while cleaning out the tip. The posterior zygomatic cells removed up to inner cranial table. Dura of middle fossa not exposed. Wound packed as usual and partial suture.

June 22d. — Patient in good condition except for intestinal distention and scanty urine. Temperature 98° , pulse varying from 56 to 80. Had $\frac{1}{4}$ gr. morphia with strychnine before operation and two injections ($\frac{1}{4}$ gr. morphia with strychnine) during the night to control restlessness. Not much pain in the head; rational; says he feels well except griping pain in the abdomen. Urine scanty (24 hours' specimen before operation showed 496 cc., 1024 sp. gr., albumen none; casts, considerable number of hyalin). After operation, no urine voided for sixteen hours, when 352 cc. of urine voided, but lost. The colon considerably distended, also the stomach. No facial paralysis.

July 13th (22d day after operation). — Patient had made an uneventful convalescence to this date. Had not had any fever; had been sitting up a part of each day on the porch. Slight headaches, but no return of the severe headaches and pain in the mastoid. His brother had gone back to his home. The mastoid was filling in clean and red. Dressed daily. Still some creamy pus discharging into the mastoid wound from the middle ear (likely due, it was thought, to the long-standing middle-ear suppuration), a few drops at a time; not enough to cause uneasiness. A small amount of pus can also be squeezed out by pressure on the neck just behind and below the mastoid.

July 17th. — Four days past a gradual and progressive change for the worse was apparent. Patient had grown weaker; become more restless and slight delirium was noticed. His temperature had varied from 99.2° to 101° the past two or three days. No paralysis and no focal symptoms were apparent. Eyes examined and showed no choking of the

disks. A marked nystagmus was seen when patient was directed to look away from the affected side, lateral in character with slight rotary motion. When eyes were directed towards the affected side no nystagmus was seen. No involvement of the third, fourth, fifth, sixth, or seventh nerves was apparent. Kernig sign, questionably present. Some pain behind the mastoid. Mastoid wound clean and nothing to account for the lighting up of symptoms that were suspicious of intracranial involvement.

July 18th.—Marked change for the worse occurred during the night. Patient delirious at intervals; answered questions when aroused. Some twitching of the muscles of both arms. No convulsions; no localizing sign or symptom. Slight fever. Pulse 110 to 120. No vomiting. Nystagmus cannot be made out. Slight stiffness of the neck. Meningeal taché marked. The diagnosis of meningitis, resulting from an unopened abscess, was made. An exploratory operation suggested as the only hope and a poor prognosis given.

July 18th.—Operation. An effort was made to find the localized collection of pus which was believed to exist from which the general meningitis was resulting. The findings at the time of the previous operation, namely, the carious defect in the inner table of the posterior fossa, exposing the sinus and cerebellum, pointed to the posterior fossa as the possible site for the pus. The cerebellum was accordingly uncovered behind and below the sinus, but no extradural collection of pus was found. The dura was normal. The cerebellum was then uncovered in front of the sinus in the angle between the sinus and the antrum and found normal. The cerebrum was uncovered above the antrum, and no extradural collection of pus found at this location, nor any signs of present or past pachy-meningitis. It was then concluded that an intradural abscess must exist from which the general infection had resulted. The cerebellum and the temporo-sphenoidal lobe of the cerebrum were explored, but no abscess found. The lateral sinus was then opened and found normal. Patient was returned to bed without the cause for the general meningitis having been found.

The patient died at 2 A.M., July 20, 1908, thirty-four hours after the operation, without any changes in the course of

the general meningitis being noticed. Autopsy was asked for and permitted, to the extent of examination of the brain.

Autopsy.—Calvarium removed; no extradural collection of pus seen. Outer surface of dura practically normal in appearance. On stripping off the dura from the cerebrum, pus found scattered all over both hemispheres. No collection at any one place except at the base of the brain. In the interpeduncular space there was sufficient pus accumulated to obscure landmarks until wiped off. The pus exudate was confined to the pia and arachnoid. On stripping off the membranes the surface of the brain was apparently not diseased. Section of the cerebrum failed to disclose any local collection of pus or signs of encephalitis. The ventricles were normal, apparently free from purulent extension. Examination of the posterior fossa showed the cerebellum to be entirely free from pus except on the left side near the anterior border, where a surface area about the size of a quarter was covered with pus. Section of the cerebellum failed to show any localized collection of pus (an abscess of the brain, not reached by operation, was diagnosed and considerable chagrin felt at not finding such). The base of the skull was then examined. It appeared apparently normal, but on stripping the periosteum from the petrous bones an abscess cavity was found at the tip of the petrous bone of the right side. (See accompanying photograph.)

The petrous bone, from the prominence of the superior semicircular canal inward, was greatly altered in appearance by reason of the necrosis that was found. At the tip was an abscess cavity about the size of an ordinary bean, in which a sequestrum was found. This abscess cavity was in immediate contact with the carotid artery. The abscess cavity had been covered by, and only discovered upon stripping off, the Gasserian ganglion. External to this abscess cavity the bone was found necrotic with here and there pus in small amounts as far as the prominence of the canal. At the site of the cochlea, the bone was softened by necrosis, and on removing with the curette this softened bone, the anatomical appearance of the cochlea could not be made out. It was believed that the cochlea was full of pus and the bone surrounding had become softened. The superior semicircular

canal was opened (the bone covering it was not necrosed). The lumen of the canal did not contain fluid pus, but was reddish in appearance. The tympanum was full of pus. It was impossible to make out the connection between the diseased tympanum and the diseased internal ear and petrous bone, due to the faulty dissection. A fistula would likely have been found had we taken the precaution to examine the tympanum first instead of following the necrotic petrous bone from within outward.

The photograph, taken before touching the petrous bone except with cotton to wipe out the pus, shows, on careful examination, the difference between the diseased and the healthy side.

BACTERIAL SUSPENSIONS IN THE TREATMENT OF AURAL SUPPURATION.¹

By RAY CONNOR, A.B., M.D., DETROIT, MICH.

THE problems of immunity are endless in their multiplicity and complexness. The forms of immunity are varied in the different ills to which flesh is heir. It is not my purpose to-night to go into a discussion of the theories either of antitoxic, bacteriolytic, or phagocytic immunity, but merely to present a few clinical observations on the use of suspensions of dead bacteria introduced into the systems of patients having localized infections in the temporal bone. While much has been written on the general subject of bacterial inoculations, still very little has appeared as to its usefulness clinically in ear conditions, and this may serve as an excuse for the cases quoted to-night. The value of the opsonic index certainly seems open to question as a guide in giving bacterial injections, and, from considerations of time, had to be omitted in these cases. Whether or not the results would have been better had it been employed, I cannot of course say.

Let us first consider the cases of suppuration treated without operation. These are all chronic cases and most of them very unpromising ones.

ANNIE T., white, æt. fifteen months, was admitted to the Children's Free Hospital, June 20, 1908, with pneumonia,

¹ Read before the Detroit Ophthalmological and Otological Club, Dec. 1, 1908.

which ran nearly two weeks before the left ear was seen to be discharging profusely. On examination the left ear showed a large round perforation behind the malleus, with no mastoid tenderness or swelling of the canal wall. This was treated by irrigations until August 6th, when a culture was taken and proven to be pure streptococcus. A vaccine was made and given in from 10-25 million doses weekly until October 29th when the discharge had completely ceased, and on November 6th the patient was discharged cured and has not been seen since. Thus a case which had resisted treatment for a month cleared up only after nearly three months' treatment with the injections.

ROY M., white, æt. four days, was admitted with an ophthalmia neonatorum. It was a malignant case. Both corneæ were badly infected, and in spite of all treatment both eyes were lost, the lens on one side being extruded. On January 4, 1908, a purulent discharge from the right ear was noted. On January 8th, the discharge was very profuse, the ear excoriated, and the canal wall swollen so much as to prevent the recognition of any landmarks in the fundus of the ear. A culture showed Friedlander's pneumobacillus. On January 11th, the patient was given ten million of the dead bacilli and six days later it was noted that the discharge from the right ear was less. There was less swelling of the canal wall, but a marked drooping of the posterior superior canal wall was still present. The left ear was now found to be discharging a thin muco-purulent secretion, and great drooping of the superior wall was found. An internal Wilde's incision was made and staphylococcus aureus isolated from the discharge. The patient was then given weekly doses of both organisms varying from 50-200 million of the staphylococcus and from 25-50 million of the bacillus. This was continued for about six weeks and showed improvement after four weeks. On March 14th, there was no discharge from either ear, the drums were gray, and the swelling of the canal had subsided. The general condition of the patient was poor and he weighed but five pounds. Local treatment was discontinued and the patient went home, where he died a few weeks later without any return of the discharge. This case is interesting in that the ear which began to discharge after

the treatment was begun showed a different organism from the first ear, and the effort to produce a relative immunity to one organism did not prevent another from getting in its work. This case was the exception to the general rule, that improvement in the aural condition is accompanied by a general improvement.

PEARLIE J., white, æt. ten months, was in the hospital for other complaints. In January, 1908, the baby had an attack of scarlet fever and when seen January 26th, the left ear was discharging freely. There was no great swelling of the canal wall and a culture showed staphylococcus aureus. A suspension was made and the baby given injections varying from 50-500 million at weekly intervals until June 1st. On March 24th, there was still a large anterior perforation, no discharge on irrigation, and only a little in the tympanum. The patient gained weight and improved generally very much. On April 23d, there was still a slight discharge from the ear, which cleared up during May and the baby was allowed home. We later heard from the mother that the ear was discharging again. The perforation never closed and the case can hardly be classed as cured.

CHLOE N., white, æt. seventeen months, was admitted March 26, 1907, in a miserable condition, from an institution in another city. Both ears were discharging profusely. The patient had a large bedsore over spine, enlarged glands in neck and groin, and a hemorrhagic petechial eruption over body. The right drum was not bulging and the canal wall was not swollen. The left canal wall was swollen, large tympanic perforation present, and the landmarks of the drum not to be made out. April 7th, a large perilaryngeal abscess was opened by Dr. B. R. Shurly. A month later the child still looked miserable and had gained little. Local treatment to ears and general supporting treatment was used during the summer without great improvement. On October 22d, a culture from the left ear showed a mixed infection of staphylococcus aureus and albus. A mixed vaccine was made from 24-hour growths, and injections given at intervals of from five to seven days and ranging in amount from 100-500 million. This was continued well into January, 1908, and the child's general condition began to pick up almost at once.

She gained in weight from 14 pounds to 21 pounds 14 ounces, and the discharge diminished steadily. On November 23d, the right ear showed no discharge, but a large perforation and some moisture in the tympanum. The left ear was dry, drum white, perforation open and showing no signs of closure. The patient showed a slight rise of temperature after a dose of 500 million, but none on dropping the dose to three hundred million. On January 8th, both ears were dry. On January 18th, the left ear dry, drum gray, and still shows a small anterior perforation unclosed. Right ear dry, drum gray and shows a large perforation of the lower part of membrane. Inner wall of tympanum not congested. The patient was discharged January 21st. About two weeks later it was reported from the Home of the Friendless that there was some aural discharge. I have not seen the case since. It is unfortunate that no cultures could be obtained from the recurrent discharge in these last two cases, as it leaves one uncertain whether the patient was suffering from a new infection with some other organism or from a lighting up of the old infection for which no effective immunity had been established.

EVA R., white, æt. five years, was readmitted on January 11, 1908. She was an old hospital case, coming in months before with typhoid. She developed mumps and then diphtheria. Then had scarlet fever and chicken-pox. The ear began to discharge on December 27th. On January 13th, the right ear showed a moderate amount of muco-purulent discharge with a foul odor. Most of the drum was gone, there was a tit-like drooping of the posterior superior canal wall, but no mastoid tenderness was present. A culture showed a mixed infection of pneumococcus and diphtheria bacillus. She was given injections at weekly intervals varying from 5-50 million diphtheria bacillus and 10-50 million pneumococcus. On February 8th, there was very little discharge in the middle ear. On February 23d, some patches developed in the throat and the patient was returned to the contagious hospital for a week. The later part of April the discharge ceased and did not start again although she was kept under treatment until the last of August. She was watched until September

24th and then allowed to go; I have not heard from the case since.

STASIA M., white, æt. 2½ years, was admitted from another institution November 6, 1907, without any history. The right ear showed a muco-purulent discharge, landmarks and most of drum gone, and granulation tissue in the middle ear. Numerous streptococcus and a few staphylococcus colonies were found in the culture taken from the discharge. She was given injections at from 5-7 days' intervals, varying from 100-500 million of the staphylococcus aureus and from 10-50 million of the streptococcus, until February 22, 1908. The discharge varied a good deal while under treatment, sometimes being very little and at other times a great deal. On January 8th, there was little discharge, but the condition of the tympanum was much the same as when beginning treatment. A culture showed abundant yellow staphylococcus. The condition of the patient remained much the same until February 22d, when a radical operation on the ear was advised and refused. The case was discharged uncured.

These cases comprise the unoperated chronic middle-ear cases that I have treated for any considerable length of time, and do not make a very brilliant showing for this method as the routine treatment in this class of cases. They all required months of treatment, one was entirely uncured, and at least two of them recurred after leaving the hospital. They were, however, very unpromising specimens and possibly private cases in good surroundings might do better.

One case was treated without material result for four months and then subjected to a radical mastoid operation. ROSABELLE M., white, æt. five years, was admitted to the hospital with cerebro-spinal meningitis from which she recovered. She had had a discharging ear since infancy. On examination a profuse muco-purulent discharge was found in the left ear. The short process of the malleus could still be seen, but the lower part of the drum was completely gone; granulation tissue was found in the tympanum, but no dead bone could be felt with the probe. There was no swelling of the

canal or tenderness over the mastoid. A culture showed one of the proteus group present, and treatment was begun on November 14th with fifty million and continued in doses running up to one billion at from five- to eight-day intervals until July 11, 1908. After the billion dose the patient felt sick, with pains in the head and back, and showed a slight rise of temperature. This passed off in a day or two and did not follow smaller doses. On January 8th, there was still a thick, purulent, bad-smelling discharge, but not enough to run out of the canal. Local conditions were unchanged and a culture still gave proteus. On May 5th, the improvement was so little that a radical mastoid was done under ether. The mastoid cortex was found greatly thickened and sclerosed. The antrum was small. Only a small part of the head of the malleus was found in the tympanum. The wound healed slowly and now shows only a little moisture in the region of the tube. Cultures taken recently showed no proteus but a few staphylococcus colonies, so that the immunity to the proteus seems permanently established. The injections which followed the operation did not, as far as one can judge, seem to influence the healing to any marked extent. Other similar cases under like conditions seem to do equally well with local treatment alone.

Several acute mastoids which have had simple mastoid operations have been treated throughout the post-operative period but without striking results.

HARRY S., white, æt. 5½ years, was admitted July 21st through the kindness of Dr. Frothingham. Patient had measles four weeks before and ears have discharged since. A swelling behind the left ear was noticed four to five days ago. This post-auricular abscess was opened under ether, and the mastoid was found sclerosed and filled with granulation tissue. The sinus but not the brain was slightly exposed at operation. The antrum was opened freely, but the tip was not completely removed. On July 24th, the wound was dressed and considerable discharge found. There was some discharge from the left ear but none from the right. On August 1st, a culture from the left side showed two staphylococcus aureus colonies and many streptococcus colonies.

On August 5th, the index to streptococcus was found to be .69 and ten million streptococcus suspension was given. On August 9th, the index was found to be .92 and there was no discharge from the canal. Injections running from ten to fifty million were given at weekly intervals until September 14th, when the boy was discharged with the wound practically well.

A case of epidural abscess which did not heal well after six weeks was given some injections of the micrococcus catarrhalis and the wound showed immediate improvement and was completely closed about three weeks later. Another similar case of epidural abscess was treated with autogenous streptococcus and staphylococcus aureus suspensions, beginning ten days after operation, but healed slowly and was not completely closed after three months' treatment. Repeated cultures gave only staphylococcus aureus. When, however, the injections were stopped, the progress of repair retrograded rapidly and the amount of secretion increased greatly. In still another simple mastoid the repair went along nicely for about two months when progress seemed to stop. A wound about 1cm long and 1cm deep was present, and while only secreting moderately still showed little tendency to close completely. A pure culture of staphylococcus albus was found and a few injections closed the wound completely and permanently in a couple of weeks.

The cases which have been longest under treatment are of course chronic ones, and in these perhaps the bacterial suspensions have been of the greatest service.

EMMANUEL R., white, æt. six years, was admitted June 2, 1906, with a chronic mastoiditis. The patient had had black diphtheria and scarlet fever two years ago at the same time, and was blind, deaf, dumb, and paralyzed for a year following this. He had received treatment in various clinics. When first seen, both drums were gone and a profuse mucopurulent discharge was present. Watch was heard only on firm contact and the voice badly at one foot. He was treated locally for a time and on September 13, 1906, a radical mastoid was performed on the left ear. The cortex was found very hard, the antrum large and filled with a mucoid

discharge, and the mastoid diseased to its tip. All diseased tissue was removed from the mastoid. The tympanum was curetted and only a little of the head of the malleus found. The wound healed poorly after operation, and the middle ear did not epidermatize, so that on January 23, 1907, the mastoid incision was reopened and the mastoid and middle ear recuretted and grafted with Thiersch grafts from the patient's thigh. The wound still refused to heal well, and on April 21st, Bier's hyperæmia treatment was applied for twenty hours out of twenty-four and continued for about four months with indifferent success. On August 9th, there was still discharge and odor from the operated side. A culture taken proved to be practically a pure culture of pyocyaneus with a few staphylococcus. On August 13th, he was given one hundred million of the pyocyaneus suspension. Four days later there was much less odor and discharge and the green color had disappeared from the dressings. Treatment, continued up to the last of October, caused the greatest improvement in the local conditions. On October 14th, cultures taken from the left ear showed no growth at the end of twenty-four hours. On October 20th, the patient was sent home to get a little change before operation on the other ear. There was a little moisture in the region of the tube, but otherwise healed and with some improvement in the hearing. One must consider the long and ineffectual treatment in this case to appreciate the value of the bacterial treatment. The right ear was operated on this year and, whether or not due to the bacterial treatment of last year, healed very much more readily than the left one.

CHARLIE H., white, æt. two years, has been in the hospital with one thing and another practically all his life. When two months old his ears began to discharge, and in spite of all kinds of treatment have continued to do so since. On May 9, 1907, a meato-mastoid operation was done under ether, with an immediate grafting of the wound on the left side. The cortex was found to be very thick and dense. The antrum was large and filled with muco-pus. The wound did not heal well after operation and the patient came down with chicken-pox. Bier's hyperæmia treatment was tried for about two months without success. On July 14th, there

was not much discharge, but considerable odor, and cultures showed colon infection. The index to colon was .9, and on July 18th ten million of a colon suspension was given. Injections were given weekly in increasing doses. On July 30th, there was still slight discharge and odor, and a culture still showed colon. On October 12th, the wound was practically healed, and a culture taken two days later showed no growth after 21 hours. On October 17th, a culture taken from the right ear showed a pure culture of *staphylococcus aureus*. Suspensions of colon and aureus were continued until January 4, 1908, when both ears began to discharge again. A culture taken from the left ear showed Friedlander's *pneumobacillus*. Suspensions of the *pneumobacillus* and aureus were given until February 22d, when the patient went home for a few days. On March 27th, a radical was done on the right side, but the middle ear still shows slight discharge. A culture recently taken from both sides showed pure *pyocyaneus* infection, which has improved greatly under injections of *pyocyaneus* suspensions. This case was, however, apparently cured permanently of a bad colon infection, but the unoperated ear was little influenced by treatment.

The patient I have had longest under continuous treatment is ERWIN E., white, æt. 3½ years, who was admitted May 30, 1905, with a diagnosis of mastoiditis. Two weeks previously he had been operated on at another hospital for adenoids. The left ear was found to be discharging profusely, with a very tender canal, marked bulging of the posterior superior wall, but no swelling or tenderness over the mastoid. The patient looked sick and miserable. An internal Wilde's incision was made and the patient put on aural irrigations. On June 4th, the right ear required an internal Wilde's incision. The patient continued to do badly, and on June 13th both mastoids were opened and found filled with pus and granulation tissue from aditus to tip. The mastoids were thoroughly cleansed out and the remains of tonsils and adenoids removed. The patient was very sick after the operation and nearly died. His general condition improved slowly, but the wounds did badly, and after long treatment the left ear was still discharging and bare bone could be felt on the left side. A radical was done

on the left ear on December 5, 1905. The old mastoid wound was found filled with exceedingly unhealthy granulation tissue and was cleaned out. Ten days later a skin grafting was done, but most of the graft sloughed out and the wound broke down. On June 30, 1906, the sinuses on both sides were discharging freely. A tuberculin test was tried but no reaction obtained. A course of antisyphilitic treatment yielded little result. On October 27, 1906, Dr. Ohlmacher got a streptococcus from the right side and an albus from the left. A streptococcus suspension was made and small doses tried for a few weeks without much permanent improvement. On April 21, 1907, Bier's hyperæmia treatment was begun and tried for ten weeks without much success. On July 6, 1907, a suspension was made from a streptococcus obtained from the right side and twelve million given. The index for the next day for streptococcus was found to be .5. On July 9th, the index had risen to .88 and two days later fallen to .6. The discharge was much less. On July 13th, given twenty million. There had been practically no discharge in the past three days. On July 14th, the index to streptococcus was .58. On July 28th, a culture from the left side showed staphylococcus albus and suspensions of this organism were given at the same time with those of the streptococcus. On July 31st, the index for staphylococcus albus was 1.1, and on August 9th the index to streptococcus was .54. The patient has been under weekly injections since this time, making something over a year's treatment. The highest dose was one hundred million streptococcus, one billion staphylococcus aureus, and three hundred million staphylococcus albus all given at the same time. The smaller doses, however, seem to do better. At present there is no discharge from the right mastoid and little from the left, which is epidermatizing slowly. It seems to be a case of very low resistance to the streptococcus, which responds but poorly to treatment.

In the many hundreds of injections given, some to miserably-nourished babies, I have never had any abscess formation or trouble at the site of inoculation. The general symptoms from the inoculations have been

marked in a few instances but have never been alarming. No case seemed harmed in any way, although in many the effect was not marked clinically.

CONCLUSIONS.

This is not a method available for routine or general use in the chronic suppurative diseases of the ear.

Those cases in which free drainage has been established by operative means are the most likely to be benefited.

Benefit may be obtained in some intractable cases which resist all other means of treatment.

Secondary operations may sometimes be avoided.

My thanks are due Parke, Davis & Co.'s research laboratory for the courtesies which made these studies possible.

REPORT OF THE MEETING OF THE AUSTRIAN OTOLOGICAL SOCIETY.

BY DR. ROBERT BÁRÁNY OF VIENNA.

Translated by Dr. GERHARD H. COCKS, New York.

MEETING OF JUNE 22, 1908.

V. URBANTSCHITSCH (1) demonstrated a case of tubercular disease of the middle ear in a patient with pulmonary tuberculosis. Before operation, hearing power for C sharp 2m, and F sharp $\frac{1}{2}$ m. The patient hears Bárány's noise apparatus without difficulty. Marked fistula symptom. Considerable rotatory and horizontal nystagmus toward the diseased side is exhibited when pressure is made over the tragus. This shows even more clearly when the air in the auditory canal is condensed. During rarefaction, nystagmus occurs to a slighter degree in the opposite direction toward the sound side. During bending and quick movements of the head there are also vertigo and nystagmus. Caloric reaction present. The diagnosis of fistula of the semicircular canal was made,—apart from the operation,—and taking into consideration the preservation of the hearing, Neumann's local anæsthesia was employed because of the pulmonary involvement. After the completion of the radical operation a broad fistula was seen in the semicircular canal. Above this fistula, opposite the middle cerebral fossa, a sequestrum was wedged in between the pyramid and the dura. When Urbantschitsch grasped this sequestrum and moved it with moderate force, the patient complained of considerable vertigo and twitching of the eyes (nystagmus). As soon as the sequestrum was removed, the patient's head felt free and the

vertigo ceased. Pressure upon the fistula with cotton did not produce vertigo. Following the operation, rotatory nystagmus toward the sound side occurred for a time, which gradually diminished in intensity. The patient is now completely deaf; the caloric excitability has greatly decreased. Spontaneous vertigo has not occurred since the removal of the sequestrum. Urbantschitsch leaves the question open whether this sequestrum pressed upon a second fistula situated in the superior semicircular canal, or whether the vertigo was caused by the pressure exerted upon the dura by the sequestrum at the moment of extraction.

Urbantschitsch (2) reported the autopsy findings in a patient who died from purulent meningitis of otitic origin. She came to the clinic with remarkably few symptoms. A chronic middle-ear suppuration was present. At operation, Urbantschitsch found the dura of the posterior and middle fossæ, together with the sinus, covered and infiltrated with pus. Because of the high temperature, Urbantschitsch incised the dura and evacuated purulent serum. Incision of the brain disclosed no pus. Completion of radical operation. On the first day after the operation the general condition was good. No headache. On the second day slight frontal headache, soon coma, and exitus. Rutin stated that there was a post-mortem rise of temperature. At the necropsy, Störck found a purulent meningitis. In the sinus petrosus superior there was a purulent thrombus. The slight clinical manifestations were a striking contrast to the fulminating meningitic process.

BÁRÁNY: Diagnosis of labyrinth fistula with demonstration. Bárány presented a patient on whom Prof. Urbantschitsch operated because of chronic suppuration and labyrinth fistula. The interesting point in connection with the case is that the patient was carefully examined clinically before the formation of the fistula. This examination was carried out by Dr. Kiproff, who is at present working upon the exact determination of caloric nystagmus. Dr. Kiproff states that caloric nystagmus, at the first examination, began 15" after irrigation with warm water at 30° Celsius, and lasted 2' 20". In the sound ear, nystagmus began after 10" and lasted 3' 5". Rotatory nystagmus toward the diseased side

lasted 55" after 10 revolutions; the rotatory nystagmus toward the sound side, 40" after 10 revolutions. Fistula symptom negative. The hearing distance for Cs, 6m; and Fs, 1m. Eight days after this examination the patient came complaining of vertigo, which began suddenly, three days before. There is considerable vertigo when walking, when making rapid movements of the head, and upon bending. When inclining the head backward a more marked nystagmus toward the diseased side is noted. There was no spontaneous alternating nystagmus, but there was slight nystagmus toward the sound or diseased side. When testing for the fistula symptom condensation of the air causes marked nystagmus toward the diseased side, and rarefaction a slighter degree of nystagmus toward the sound side. The caloric test shows nystagmus begins after 30" when syringing with water at 30° C. and lasts 3' 20". In the sound ear, it begins after 15" and lasts 2' 50". The duration of rotatory nystagmus is 40" to the diseased and 45" to the sound side. In a second examination, three days later, the caloric nystagmus in the diseased ear begins after 10", lasts 3' 10"; while in the sound ear it begins after 15" and persists 2' 55". Rotatory nystagmus toward diseased side 1'; toward sound side 45". Fistula symptom same as before. Hearing power same as at first examination. At the radical operation, a distinct fistula was found in the semicircular canal. After operation, the nystagmus toward the sound side ceased, the hearing power was lowered considerably, and the irritability of the vestibular apparatus was substantially diminished. This report is of great interest because it is the first case where an exact functional examination of both cochlear and vestibular apparatus was made before a fistula originated spontaneously. This case seems to indicate that an abnormal irritability of the vestibular apparatus was caused by the formation of a fistula, which manifested itself in the occurrence of attacks of vertigo (spontaneously and with movements of the head). However, a hypersensibility of the vestibular apparatus, as Alexander has already assumed, was not present; for the figures obtained by Dr. Kiproff agree fully with average figures in middle-ear suppurations having a similar anatomical character. Whether there is actually a physiological hyper-

susceptibility by which the vestibular apparatus responds to physiological irritation by increased function, appears to Bárány doubtful.

In connection with this case Bárány reported his experience during the last few months in the University Ear Clinic of Professor Urbantschitsch, in connection with the fistula symptom. Altogether 160 patients were examined for fistula before operation. Of these, 145 cases exhibited no fistula symptom, and no fistula was found at operation. Bárány emphasized the fact that he knew of no case, carefully examined before operation and showing no fistula symptom, where a fistula was found at operation. All of these cases also showed typical caloric excitability. In 4 cases where there was no fistula symptom, caloric excitability was negative. In all of those cases where the diagnosis of labyrinthine suppuration was made at operation, the fistula was found and the labyrinth operation performed. In 11 cases the fistula symptom was present before operation. So far, 9 of these have been operated upon. In all, the fistula was situated in the semicircular canal. Six cases showed a pronounced fistula symptom, also marked nystagmus during both condensation and rarefaction. In these cases the caloric excitability was normal; in some the hearing power was present, in others deafness. In three, the fistula symptom was very slight; in place of nystagmus there was only a very slight slow rotation of the bulb, but always in an opposite direction during condensation to the motion during rarefaction. In all, caloric excitability was either greatly reduced or abolished, and deafness present. In our hands, then, the fistula symptom has proved completely trustworthy. It should be noted that we have, by chance, only happened to observe semicircular-canal fistulæ. In most of the cases the nystagmus was in a direction toward the diseased side when the air was condensed, yet Bárány has already seen exceptions in semicircular-canal fistulæ.

In 1906, Gradenigo reported at the Tenth Congress of the Societa Italiana di Laryngologia, Otologia, et Rhinologia the investigations made in his clinic by Dr. Mimidian. The latter tested a number of cases by air condensation and rarefaction. He observed a nystagmus which occurred in

the same way in rarefaction and condensation, and which was seen most clearly in alternating rarefaction and condensation. Gradenigo did not draw any further conclusions from these findings. His observations induced Bárány to try systematically the fistula symptom. Bárány stated that where air condensation generally produced a nystagmus, rarefaction always caused nystagmus in the opposite direction. Dr. Mimidian's experience may depend upon a change in the caloric nystagmus, since the condensed air is not completely shut off, and repeated condensations and rarefactions finally produce a caloric nystagmus through the current of air.

In January, 1908, in the *Wiener klinische Rundschau*, xxii., Nos. 1 and 2, is a work by Alexander and Lassalle, entitled "A Clinic on Labyrinthine Nystagmus." In the April *Monatsschrift f. Ohrenhkk.*, an attack by Mackenzie appeared in which a number of Alexander's newly-published cases are cited and two more cases added. Alexander alleges that he has found a positive fistula symptom in all of these cases, but no fistula. Alexander's cases may be divided into three groups:

1. Cases which from the history show with great probability that there is a fistula of the stapes or round window.

2. Cases which from the description of the fistula symptom show with great probability that there is a fistula of the stapes or round window.

3. Cases which from the post-operative course show with great probability that there is a fistula of the stapes or round window.

CASE I.—Before operation, vertigo repeatedly. The test for the fistula symptom showed distinct rotatory nystagmus toward the diseased side during condensation, and the same toward the sound side during rarefaction. Symptoms of acute labyrinthitis appeared immediately after operation.

CASE 10.—Thirteen-year-old boy with bilateral chronic suppuration. With strong condensation in right canal, rotatory nystagmus to the right; with slighter condensation in left canal, stronger rotatory nystagmus to the left. No mention of nystagmus with aspiration. The disproportion

between the nystagmus on the right and left sides is best explained by a left-sided stapes fistula.

CASE II.—Before and after operation, attacks of vertigo. Facial paralysis. During condensation in diseased ear rotatory nystagmus toward the diseased side.

In all these cases, as Alexander found no fistula of the semicircular canal, we must accept a stapes fistula. In any case the opposite view, that none existed, cannot be proven. There still remain three cases in which Alexander obtained rotatory nystagmus toward the sound side during condensation. In one operative case there was no vertigo, either before or after operation.

In two cases in which the perforation in the drum closed in the course of treatment, there was no longer any nystagmus upon compression after the closure of the perforation. Bárány thought that there was a change in the caloric symptoms, due to air being forced repeatedly into the external canal, just as in rare cases a caloric nystagmus is caused by air being forced into the tube.

Finally, Bárány cited a case where Leidler confused the fistula symptom with the associated Stransky's nystagmus. The case in question was presented at the last meeting. Leidler proposed incision of the dura. In the presentation of this case Leidler did not mention compression nystagmus, otherwise Bárány would have answered him at the time. It was noted in the history sent to Bárány that compression on both ears started nystagmus. Bárány accidentally had an opportunity to examine this patient two or three days before he was admitted to the Policlinic, and stated he showed the associated Stransky's nystagmus very markedly. Bárány demonstrated to his hearers how easy in this case it is to confuse this nystagmus with the fistula symptom. Here there occurred, not only in air condensation and rarefaction, but also in pressure on the mastoid, on the tragus, and when tightly closing the eyes—a tolerably strong oscillatory nystagmus with simultaneous movements of the lids. That so practiced an observer as Leidler could be mistaken, proves how difficult it is to make accurate and exact observations in this field.

In Alexander's cases, he speaks mostly of nystagmus during

compression alone. But, according to Bárány's meaning, a fistula may only be diagnosticated if, during compression, nystagmus or vestibular eye movements are perceivable in one direction; during aspiration, in the opposite direction. In this connection Bárány desired to make an observation. Aspiration should follow immediately after compression, and the olive tip should not be removed from the ear after compression, and then the balloon compressed so that aspiration can be tried independently of compression; for condensation forces the movable parts into the fistula and interior of the labyrinth, thereby producing a movement of the endolymph. Aspiration should be performed at once, then the opposite endolymphatic movement occurs immediately, and the soft structures in the fistula are sucked at once into their original position or are drawn somewhat farther outward. If the olive is removed after compression, the soft material in the labyrinthine fistula gradually flows back spontaneously into its original position, and when aspiration is finally carried out much greater force must be employed to produce motion of the endolymph.

Bárány, in conclusion, desired to mention a point which Alexander alludes to in his paper. In twenty normal cases Alexander was never able to evoke the fistula symptom, yet he states that formerly when making Gellé's test he met with occasional complaints of vertigo. His daily notes read: "In Gellé's experiment in normal cases vertigo appears to occur if the external auditory canal is not closed in an air-tight manner. The air flowing out with a hissing sound, and the movements which in such cases the patient makes with his head may explain the statement that vertigo is experienced." From this remark, Alexander goes on to say that he first of all performed Gellé's test incorrectly, for if the external auditory canal is not stopped in an air-tight fashion it is not Gellé's test, but an inflation, and as a matter of course the patient hears nothing. Moreover, it is readily conceivable that in repeated examinations of this sort caloric nystagmus may have occurred.

Bárány next spoke of a paper by Jansen. In the Fourteenth Report of the American Laryngological, Rhinological, and Otological Society, Pittsburg, May 28, 29, and 30, 1908,

Jansen read a paper upon "The Treatment of Purulent Suppurations of the Labyrinth, on the Basis of Fifteen Years' Experience." Jansen has had an extraordinarily large amount of material, but the majority of his cases were not examined in a manner free from objection. Of late Jansen has employed the caloric test of the vestibular apparatus, but he does not speak of the fistula symptom. At least, Bárány found no mention of it in his work. Stapes luxation plays a great rôle in his paper. If the description of these cases is compared with the cases of so-called serous labyrinthitis of Alexander and Voss, one finds a certain similarity. In these cases nystagmus and vertigo do not begin immediately following injury, but 12 or 24 hours after it, or even later. Jansen has observed 19 examples, of which only 2 healed without a labyrinth operation. Stapes luxation happened only 6 times during radical operation, 12 times during the after-treatment,—notably during curettage. The labyrinth operation was performed 13 times; in 9, healing took place. The principal distinction between Jansen's and Alexander's cases consists in the former's high mortality, while the cases of Alexander and Voss all healed. Jansen states correctly that a caloric reaction will still be obtained on the first and second days, and also that the caloric test may not prove with certainty suppuration of the labyrinth. Bárány believes that the fistula test should be performed to distinguish cases of stapes luxation from serous meningitis, whether it be with the help of a sterilizable rubber-ball which can be placed over the entire wound area and fitted in an air-tight fashion, or whether it be with the help of a pledget of cotton which is carefully pressed against the region of the stapes. If no fistula symptom exists before operation, and a fistula symptom can now be demonstrated from the maintenance or lowering of caloric irritability, then we are not dealing with a serous labyrinthitis but with stapes luxation. If the fistula symptom does not show preserved caloric excitability, a serous labyrinthitis can then be diagnosticated. This differentiation is of great importance from a practical standpoint. If we can diagnosticate serous labyrinthitis from the presence of caloric excitability, then we do not disturb the labyrinth; but if we diagnosticate stapes luxation, the labyrinth operation

must be performed at once. A serous labyrinthitis which has led to destruction of the hearing and loss of excitability in the vestibular apparatus cannot be differentiated from a purulent labyrinthitis with the same functional findings. It would be better for the patient if in these cases we perform the labyrinth operation.

Discussion.—NEUMANN thinks serous labyrinthitis can be diagnosed from purulent labyrinthitis by testing the hearing power. If speech is no longer heard in the diseased ear, but the tuning-fork is still heard through air, or at least through bone conduction, he diagnoses serous labyrinthitis. Jansen speaks in his article of 15 years' experience with suppurations of the labyrinth. He makes very incomplete hearing tests, and has employed the test for caloric nystagmus the first time but a year ago. His statements are based on material only imperfectly observed.

ALEXANDER remarks that he always examines with compression and aspiration. He believes he can prevent confusion when dealing with caloric nystagmus. Whatever the connection is between vertigo and Gellé's test in normal cases, he thinks we are not dealing here with vertigo but merely with an unpleasant feeling in the ear, as is always the case when air is forced into the ear in this way. With regard to the differential diagnosis between serous labyrinthitis and luxation of the stapes, Alexander believes the most important distinction is the appearance of the symptoms of the former after two or three days, while in stapes luxation the symptoms come on more quickly.

RUTTIN has examined histologically many cases of labyrinth suppuration. Stapes luxation is very rare. Serous labyrinthitis is well established. According to his views Jansen's statistics contain many errors.

HAMMERSCHLAG differs from Neumann's opinion that hearing tests are of value in the differential diagnosis between serous and purulent labyrinthitis, and especially against Neumann's assertion that the test for bone conduction in the diseased ear is of value. Patients with exfoliated cochleæ say they hear the tuning-fork in the diseased ear if it is applied to the mastoid process on that side. The numerous examples of deaf-and-dumb people with hearing remnants

after meningitis prove that hearing remnants may remain in suppurative labyrinthitis.

BÁRÁNY remarked in opposition to Neumann that preserved hearing proves nothing in regard to labyrinthitis in the early stage, as a case he had observed with Neumann showed—which Neumann has apparently forgotten for the moment. He referred to a patient who developed an acute labyrinthitis and meningitis after an ossiculectomy, which ended fatally in two days. On the first day the hearing power for Cs was +6m. After twelve hours this was followed by total deafness. The examination with the noise apparatus proves to Bárány that the hearing power of the tuning-fork on the mastoid process of the diseased side in unilateral deafness is only apparent, because when the noise apparatus is placed in the normal ear, the strongly vibrating tuning-fork is no longer heard on the mastoid process of the diseased ear.

ALT presented (1) a 12-year-old boy, upon whom in the surgical clinic a simple mastoid operation was performed; developed facial paralysis after operation. On examination, Alt found a retro-articular opening, profuse otorrhœa, and necrotic bone in the mastoid process. After the operation, which demonstrated complete destruction of the mastoid process, the facial paralysis did not disappear. Conservative treatment for three months was without effect. Alt finally decided to operate again, and exposed the facial, which was pressed upon by a sequestrum. Since then the facial paralysis has cleared up completely.

(2) A child upon whom four and one-half years ago a simple mastoid operation was performed, and who has had a facial paralysis since that time. Otoscopic examination showed a completely epidermised tympanic cavity and slight suppuration coming from the antrum. Alt performed the radical mastoid operation, and exposed the facial throughout its entire course, from the knee above the oval window down to the tip of the mastoid process. Above the oval window it was enclosed in a tight connective-tissue scar. There was no loss of continuity. Fourteen days later there was an improvement in the paralytic symptoms, and after five weeks' active movements in the frontalis and orbicularis.

The patient was then transferred to the medical clinic for massage and electricity. The daily condition gradually became worse than it was five weeks after the operation. Now there is only slight movement of the frontalis and orbicularis, and almost no movement of the mouth. Alt suspects that fresh scar tissue may have formed. He believes that eventually the facial should be divided and embedded in a protective tube, according to Foramitti's method, to guard it against scar tissue.

Discussion.—BONDY presented a patient whose antrum he had opened on account of acute mastoiditis. For the first few days after operation the general condition was good; then the patient had a chill. Upon re-examination the wound was found covered with pus, and the sinus discolored with pus. Ligation of jugular, exposure and opening of sinus. He found a mural thrombus. Bondy excised the affected part of the sinus wall, Hofer sectioned it. The microscopic examination showed very plainly the thrombus. Aspiration would naturally have been of no use. This case proves the superiority of incision over puncture.

RUTTIN demonstrated a microscopic preparation of the Eustachian tube from a tuberculous patient. The epithelium of the tube has suppurated in part. This is of interest because it has hitherto been believed that in the acute otitis of the terminal stage of tuberculosis the infection originates by spreading mechanically. This case proves that the involvement was brought about by the suppurative inflammation of the tube.

BÁRÁNY demonstrated a patient with sinus thrombosis, who complained of earache after influenza, but there was no discharge. Fourteen days later pyæmic fever, swelling, and pain in the right ear developed. Otoscopic examination furnished the picture of an acute otitis in process of subsiding. There was no tenderness of the mastoid. On account of the pyæmic fever Bárány opened the mastoid. He found an unusually thick corticalis, and two or three cells in the antrum filled with pus. With the first few blows of the chisel, the sinus, which was situated very far forward, was exposed. After opening the antrum, Bárány followed the sinus backward. This tore and emptied out pus. Ligation of the

jugular and exposure of sinus to the region of the bulb and 3cm above the knee, enucleation of the entire thrombus. No metastases since operation. Gradual subsidence of fever.

REPORT OF THE TRANSACTIONS OF THE CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

BY DR. GEO. E. SHAMBAUGH, SECRETARY.

MEETING OF OCTOBER 13, 1908. PRESIDENT A. H. ANDREWS
IN THE CHAIR.

Dr. J. C. BECK exhibited a case of a young child with a **cyst of the epiglottis**. The case had been shown before the Society early in the year, and reported as probably cured by opening the cyst and cauterizing the cavity with electro-cautery. At the present time the cyst has returned and is already causing considerable interference with respiration. Dr. Beck expects soon to undertake to remove the cyst in its entirety.

Dr. BECK exhibited a case of a boy four years old, from whose **left bronchus he removed a small electric lamp**. The foreign body was located very distinctly by means of the X-ray. The bronchoscope was introduced, and the foreign body partially dislodged with the forceps. The patient then expelled the foreign body in a fit of coughing. Dr. Beck questions whether glass foreign bodies could always be depended upon to produce an X-ray shadow. He thinks the reason the outlines of the glass lamp showed so distinctly in this case was due to the use of lead in the manufacture of the glass.

Dr. ANDREWS has seen a number of cases where pieces of glass have produced a shadow under the X-ray.

Dr. BECK exhibited photographs of a nurse who in attempting to improve imaginary defects in her appearance, had **injected large quantities of paraffin**. The result was a

very marked disfigurement. Dr. Beck had succeeded in removing some of the paraffin surgically, as well as parts of the inflamed skin covering the masses. This tissue under the microscope presents much the appearance of a sarcoma, and Dr. Beck called attention to the supposed danger of malignancy from the use of paraffin injections.

Dr. HOLINGER reports a case in which paraffin had been injected for suppuration of the lachrymal tract. The paraffin in this case had miscarried and gone into the lower eyelid, causing an ugly irregular tumor.

Dr. ANDREWS asks if Dr. Beck knows of any reports in the literature that are authentic showing malignancy as the result of paraffin injections. He has seen none in the literature.

Dr. HEAD has incised paraffin tumors, and had little difficulty in squeezing out the masses with practically no scar resulting. He has watched the literature rather closely for a number of years, and has seen no cases reported of malignant generation.

Dr. BECK states that the particular object in presenting the specimen of the paraffinoma was to bring out this particular point regarding malignancy. Kirchner has written more on this subject, perhaps, than anyone else, and has seen spindle-cell formations about the paraffinoma similar to the case which Dr. Beck has just reported. A foreign body always becomes encapsulated, but if paraffin is the foreign body, the connective tissue also traverses the masses of paraffin, giving an appearance not unlike lipoma. There have apparently been no post-mortem reports, but Kirchner has reported cases of metastases. Only one death has followed the use of paraffin that has been reported, and that was a case reported by Pfannenstiel, who had injected paraffin for incontinence in the rectal sphincter.

Dr. HOLINGER reported a case of **extensive abscess involving the faucial tonsils**, the tissues of the neck, and the mastoid process. It did not appear to be a case of Bezold mastoiditis. The mastoid was very much sclerosed—so much so that a number of gouges were broken in chiselling away the process. This he attributed not only to the hardness of the bone, but to the fact that he used a lead mallet.

In syringing the mastoid opening after the operation, so much water would pass into the throat that Dr. Holinger believed a communication other than the Eustachian tube existed.

Dr. ANDREWS asked Dr. Holinger why he held the lead hammer responsible for the breaking of the gouges. He had never discovered that chisels were more likely to break under the lead hammer than under any other.

Dr. HOLINGER presented a microscopic section from a **postnasal growth**, which he believes showed the existence of tuberculosis. Clinically the case presented symptoms of an enlargement of the pharyngeal tonsil, which was covered with a thick muco-purulent secretion.

Dr. BECK states that evidences of tuberculosis have frequently been detected in adenoid growths, and is of the impression that tubercular nodules would be frequently found if we would examine all adenoids removed.

The section which Dr. Holinger exhibits he thinks shows an inflammatory swelling composed of small round cells, probably tuberculoma. The long duration of the symptoms would exclude sarcoma.

Dr. HOLINGER (closing) said that there was considerable difficulty in finding anything more than the diffuse round-cell infiltration, but that several giant cells had been found, which convinced him that the trouble was tuberculous. A large number of eosinophile cells, he thinks, indicates the serious nature of the trouble, and also that it has been one of long standing.

Dr. SHAMBAUGH reported a case of **cholesteatoma causing an erosion into the horizontal semicircular canal**, and discussed briefly the **methods for examining the vestibular apparatus**. The case was that of a man fifty-nine years old, who eleven years previous had a purulent discharge from the right ear, which lasted for several weeks. For several years past he has been annoyed with an accumulation in the canal of the right ear, which he supposed was caused by wax. In attempting to remove this with a hair pin he had set up a marked dizziness and nausea. The mass proved to be a cholesteatoma protruding into the meatus from an opening in the upper posterior wall, which led directly into the antrum.

The membrana tympani showed maceration and thickening, but did not appear to be retracted or perforated. There was a quick response upon syringing the ear with water only slightly colder or warmer than the body temperature. This response was in the character of a nystagmus and pronounced vertigo. On compressing or rarefying the air in the external meatus by means of the Siegel speculum, even the slightest change in pressure resulted in nystagmus and vertigo. The functional examination showed considerable deafness, more marked for the lower tones, with only a slight defect for the highest notes of the Galton whistle. The Weber was distinctly lateralized to the affected ear, and the Rinne was negative.

From these symptoms the diagnosis of an erosion into the horizontal semicircular canal was made, the erosion having made an opening only through the bony capsule, but not through the endosteum. The slight amount of pressure necessary to produce the nystagmus and vertigo in this case shows how very delicate and sensitive is the adjustment of the terminal apparatus in the ampullæ.

Dr. SHAMBAUGH discussed three methods of testing the excitability of the vestibular apparatus. All of the methods are dependent upon producing a motion in the endolymph of the semicircular canals. In the first method, that of the rotating chair, if the semicircular canals are normal and the individual is rotated toward the right, for example, there will be produced a nystagmus toward the right. If the rotation is kept up long enough, all sense of turning, as well as the nystagmus, will disappear. If the rotation is suddenly stopped, a sense of vertigo is produced, and there will be a return of the nystagmus, but directed toward the left. These phenomena can readily be accounted for by the production of a motion in the endolymph of the horizontal semicircular canals. The reason why the sense of turning and the nystagmus disappear after the rotation is discontinued for some time, is explained by the fact that the fluid in the semicircular canals has taken on the motion of the walls of the canal, and therefore does not impinge upon the cupula in the crista ampullaris. The production of vertigo, and of the nystagmus directed towards the opposite side upon suddenly stopping

the turning, is explained by the continuation of the flow in the endolymph of the canals for some time after the turning has been stopped.

The second method of producing a flow in the endolymph of the semicircular canals was the one used in this case in demonstrating the erosion into the horizontal canal. It consists in the production of either pressure or suction of the air in the external meatus. If an opening exists through the bony capsule of the canal, the endolymph will be caused to flow in this or that direction, depending upon whether suction or pressure is used.

The third method of testing the semicircular canals is that elaborated by Bárány, and consists of syringing the ear with hot or cold water. If the vestibular apparatus is intact, symptoms of vertigo and nystagmus can be produced by the use of either hot or cold water. In a case where the semicircular canals are not affected, the reactions obtained are as follows: Syringing with cold water produces a nystagmus which is more pronounced when the eyes are directed towards the opposite side. Syringing with warm water will produce a nystagmus more pronounced when the eyes are directed toward the same side. The vestibular apparatus may be placed in a state of hyperexcitability as the result of irritation, such as may occur from circumscribed suppuration of the labyrinth. Under these circumstances the normal reactions upon syringing with hot and cold water can be obtained. In addition, however, there will frequently be present a spontaneous nystagmus directed toward the affected side.

When the vestibular apparatus is destroyed, no reactions can be obtained by any of the methods for stimulating this part of the internal ear. If the destruction of the nerve endings in the internal ear has been a sudden one, there will be present for a short time a nystagmus toward the normal ear, which has its origin in this ear, and not in the affected one. Disease of the cerebellum, such as tumor or cerebellar abscess, may produce the same symptoms as occur from irritation of the nerve endings in the vestibule—that is, a spontaneous nystagmus directed toward the affected side. One can readily see how this study of nystagmus, both spontaneous and induced, is of great value in the diagnosis not

only of diseases of the labyrinth, but of such conditions as cerebellar abscess and cerebellar tumor.

Dr. HOLINGER questions whether it is feasible to apply the rotating chair examination in the office, since the space will often not permit of this apparatus. He thinks that all of these methods can be best applied in a hospital. He has had some unpleasant experiences in the use of hot and cold water in his office on account of the very severe reactions obtained. He finds that the use of water not too cold or too warm, is necessary if one would avoid an unpleasant severe reaction.

Dr. BECK thinks that Dr. Holinger's remarks might be taken as underestimating the value of the methods for testing the semicircular canals. He thinks the hot and cold water syringing of the ear in the diagnosis of labyrinthine diseases is one of the best things we have learned in otology in recent years. He has used the method in his office to his entire satisfaction. He, of course, would never inject cold water into a suppurative ear except for diagnostic purposes.

Dr. ALLPORT thinks the method of syringing with hot and cold water is our most valuable means of diagnosing diseases of the labyrinth. He has recently observed these tests applied in the clinics in Vienna, where great confidence is placed in their significance. He thinks the objections that were mentioned by Dr. Holinger are after all unimportant and do not detract seriously from the value of the tests.

Dr. ANDREWS has been making some observations regarding vertigo, and believes that we have many things still to learn on this subject. He has experimented with moving objects before the eyes, and finds that dizziness can be produced very readily. This is in accord with Deiters's theory, which locates the centre of equilibrium in the floor of the fourth ventricle. This centre, according to Deiters, receives filaments from the eye, from the ear, and from the gastrointestinal tract, and as a result an irritation from any of these localities may produce vertigo.

Fig. 1.



Fig. 2.

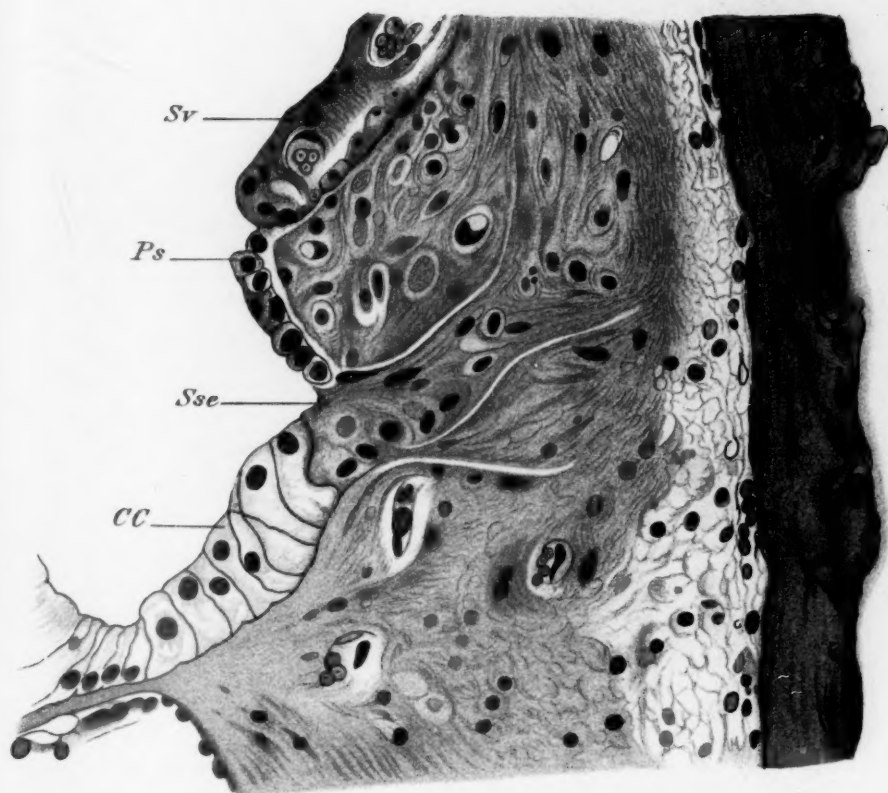
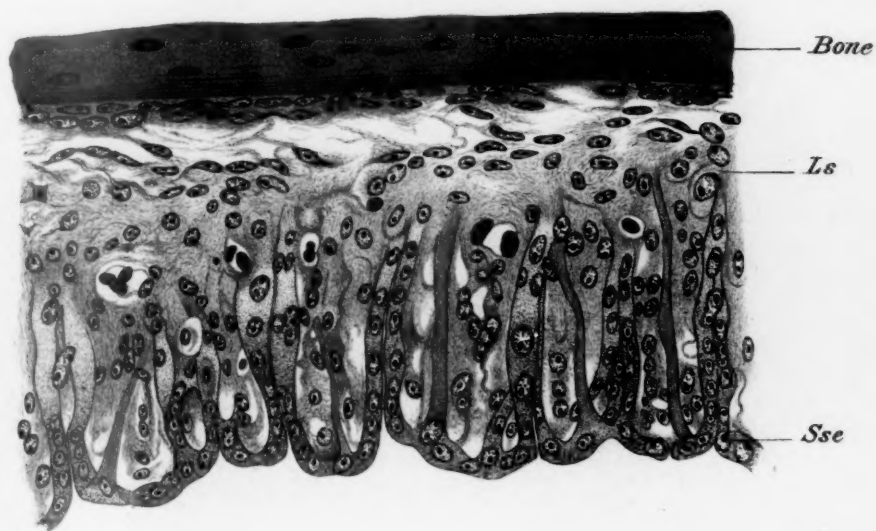


Fig. 4.



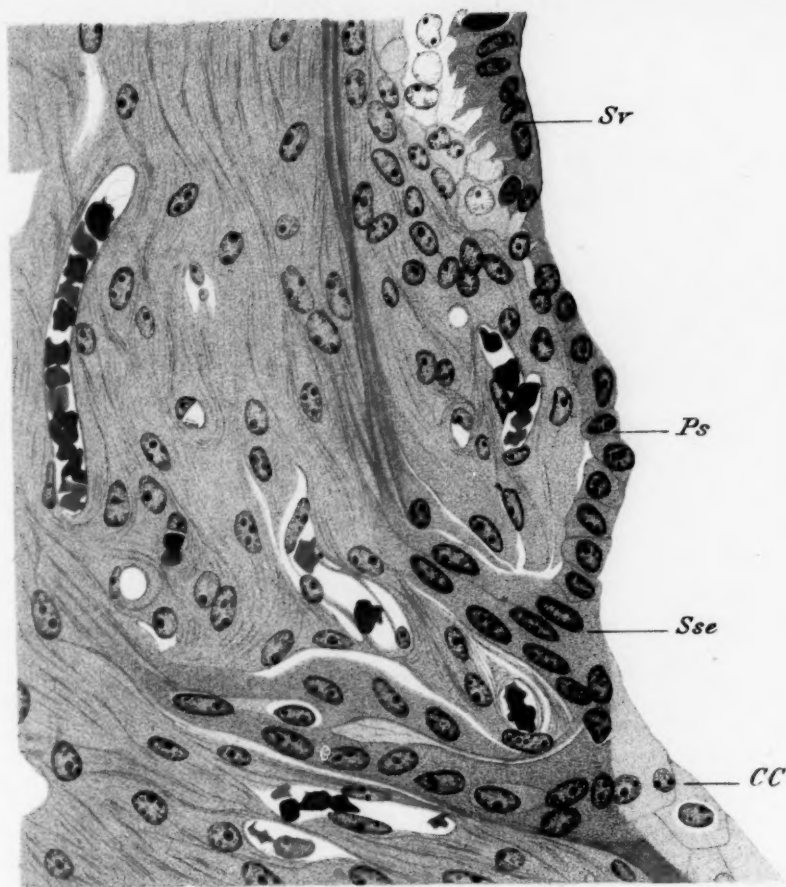
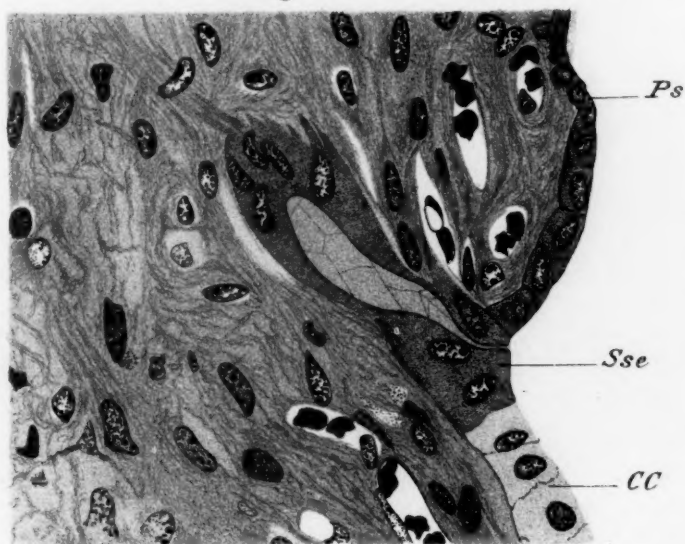


Fig. 5.



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Fig. 6.

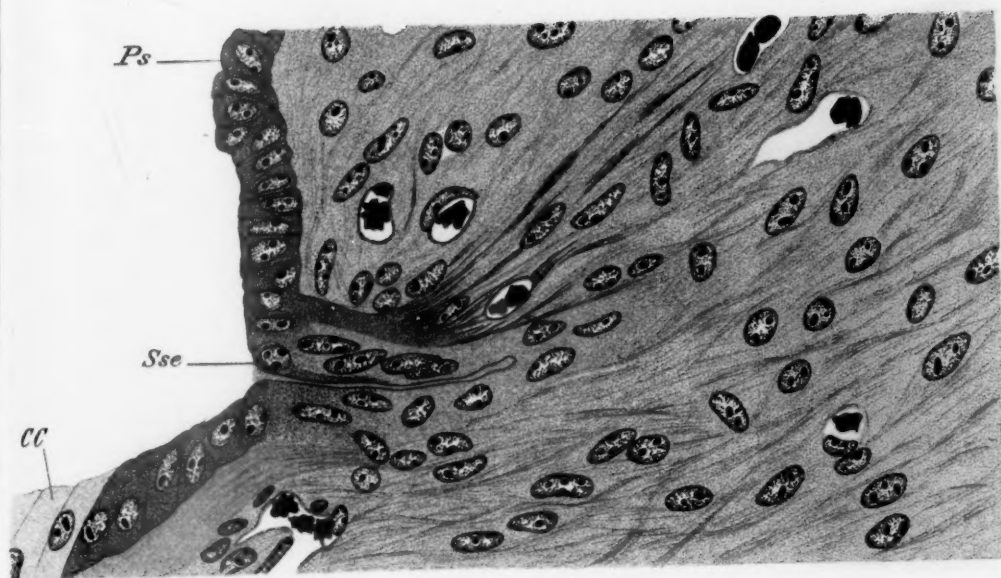
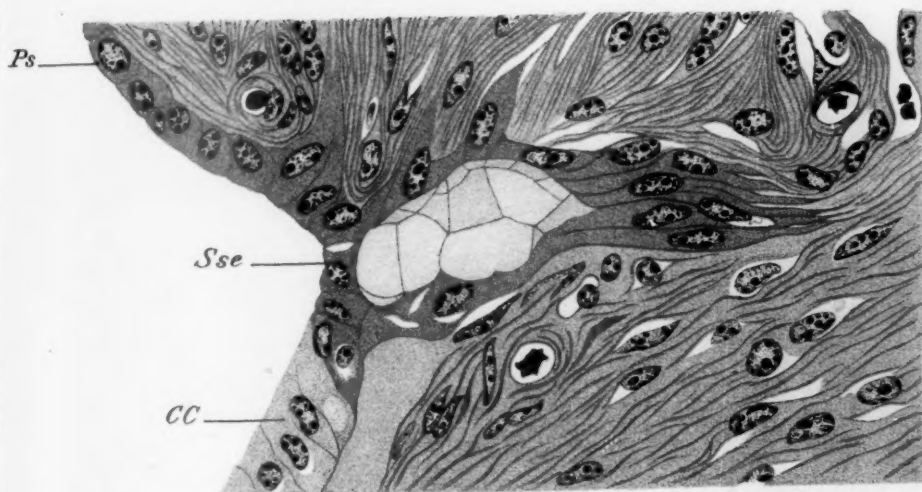


Fig. 7.



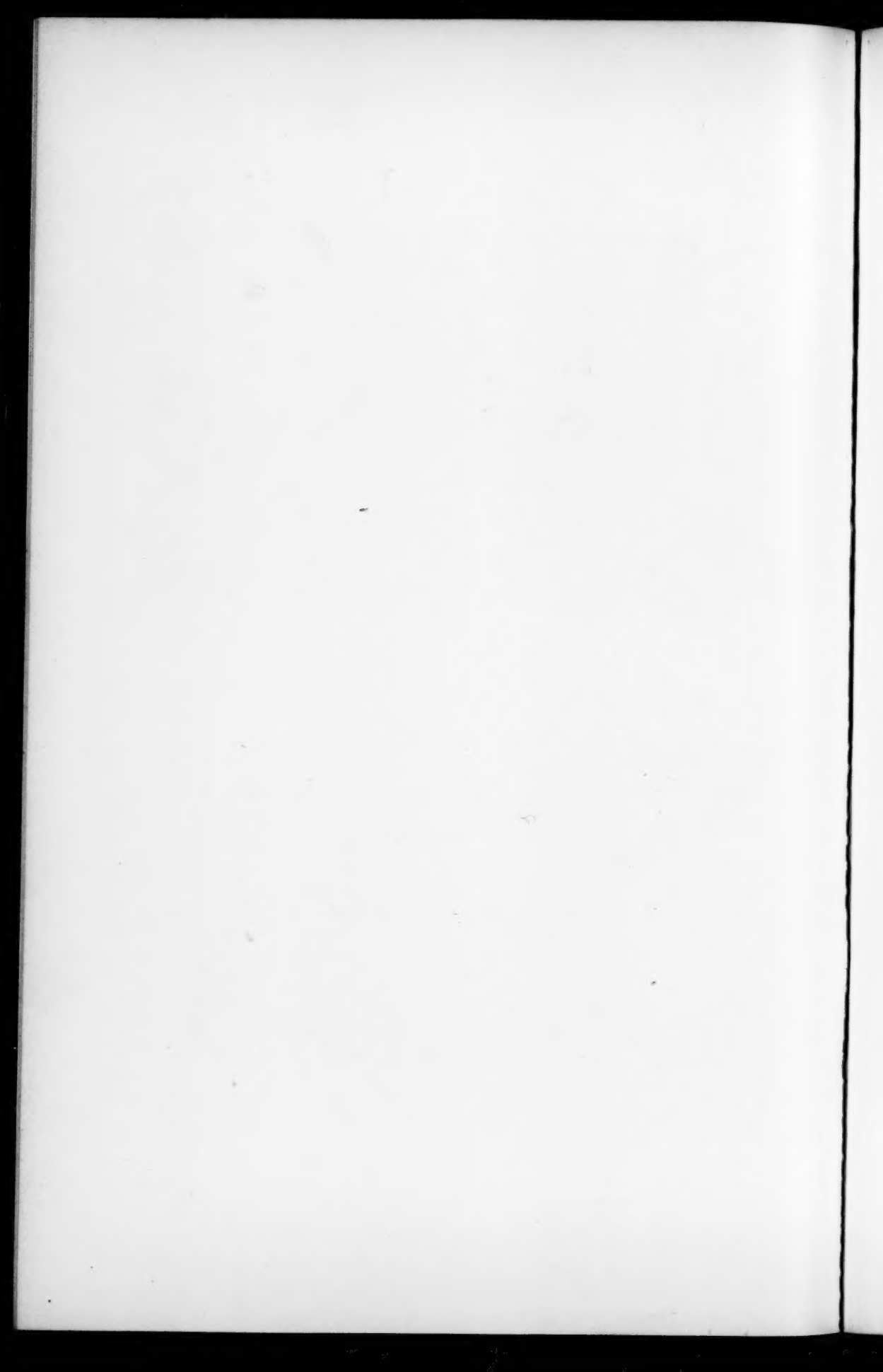
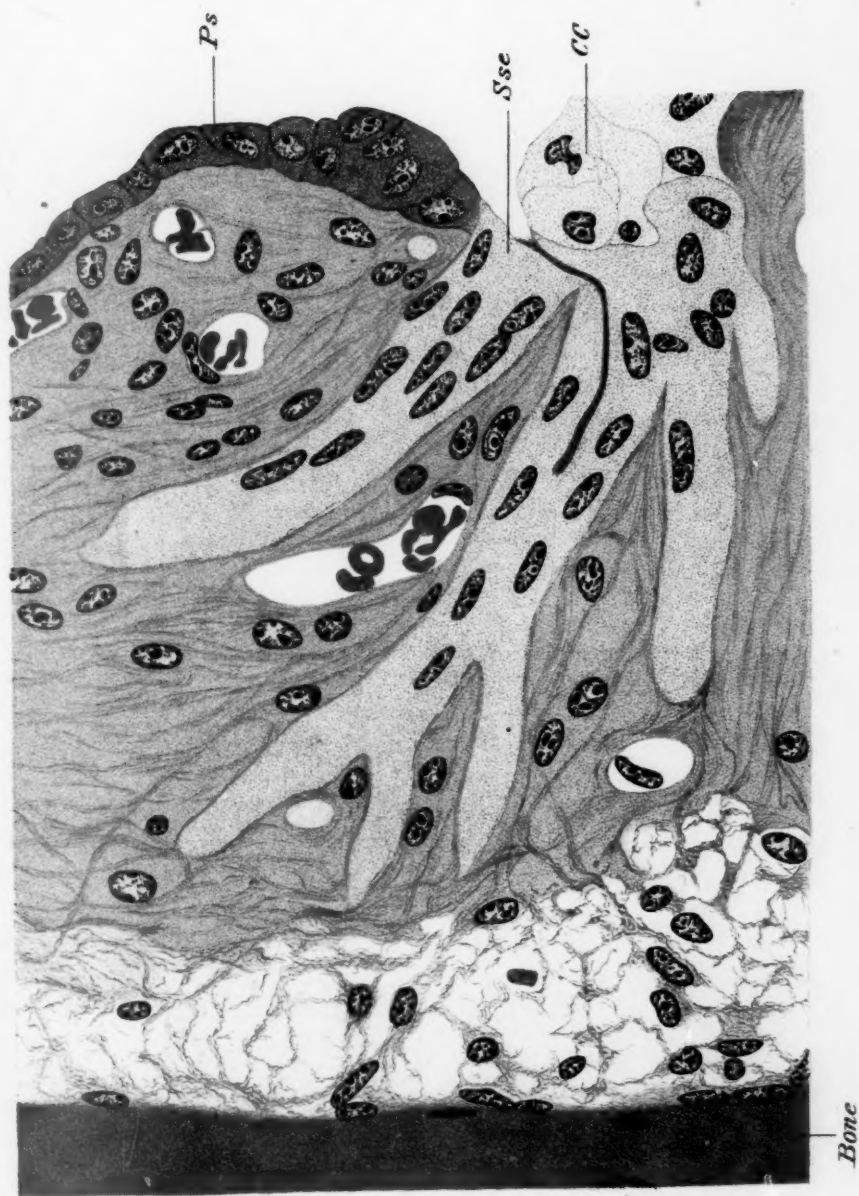


Fig. 8.



REPORT OF THE TRANSACTIONS OF THE SECTION ON OTOTOLOGY, NEW YORK ACADEMY OF MEDICINE

MEETING OF OCTOBER 9, 1908. DR. ARTHUR B. DUEL
CHAIRMAN.

Report of two cases of atypical sinus thrombosis. S. J. KOPETZKY, M.D. (Published in full on pp. 552-557 of this issue.)

Discussion.—Dr. DENCH inquired whether a bacteriological examination of the thrombus had been made in these two cases.

Dr. KOPETZKY replied that unfortunately the pathologist lost the culture in the second case, but from the microscopical appearance his opinion was as stated in the report. The first case, the one that recovered, was not examined bacteriologically.

Dr. DENCH said that in certain cases an aseptic clot was found either in the sinus or jugular vein. An aseptic clot would give rise to no symptoms. In some cases where symptoms were present, the centre of the clot might be perfectly sterile, and either extremity of the clot or the walls of the sinus or vein might contain pathogenic organisms. In one case of sinus thrombosis, in which the thrombus extended into the jugular, he had found the clot perfectly sterile, but the walls of the vein contained streptococci. In cases running an atypical course, it would be interesting to know whether we were dealing with a sterile thrombosis, and it is a question whether an aseptic thrombosis would necessarily prove fatal.

Dr. GRUENING said that in the absence of a pathological examination and the absence of a culture he did not think we were prepared to say what we had to deal with in these

cases. It was also important that the temperature should be taken every three hours, as if the intervals were longer there might be very great variations which were not noted.

Dr. KOPETZKY replied that the temperature had been noted every three hours.

Dr. BERENS said that the second case might be explained in part by the mechanical disintegration of the clot resulting from the use of the mallet and the clot acting as a thrombus.

Dr. DUEL inquired whether the mallet and chisel had been used in the mastoid operation. On receiving a reply in the affirmative, he said that the resulting concussion might have had some influence in the disintegration of the clot on the opposite side, as the reader had suggested.

During the past two years he had endeavored, in his operations on the mastoid, to use the mallet and chisel no more than was absolutely necessary, usually not at all, but to confine himself to the rongeur forceps, hand gouges, and curettes. In all radical operations as well as in acute cases he avoided the use of the mallet and chisel for the same reason.

Danger of the disintegration of a possible thrombus, a possible labyrinthine suppuration, the breaking down of nature's wall in some possible intracranial abscess made it very important that any concussion from the use of the mallet should be avoided.

Dr. HELD said that he was not sure that the chisel was entirely the cause of the trouble in this case, in view of cases which he had reported three or four years ago; but he held that the chiselling was an important factor, as the traumatism from its use had even created psychic disturbances. At the time of his report he had found nothing on the subject excepting an article by Grossman, who reported six or seven cases of psychic disturbances following the radical mastoid operation. Dr. Held spoke of a patient of his who was confined to an institution for three months following the radical mastoid operation for the reason of an acute dementia following the operation, and he believed that the use of the hammer and chisel was the cause. In the second case presented by Dr. Kopetzky he believed that the Doctor was right in ascribing the dissemination of the fatty embolism to the trauma of the hammer and chisel.

Dr. DENCH inquired whether the mental disturbance might not occur in the same proportion of cases following any severe operation upon the long bones. He remembered a case of mental disturbance which followed drainage of the ankle joint. Here, the concussion incident upon the use of the mallet and chisel certainly could not be considered as a causative factor. In over one thousand operations upon the mastoid, in which the mallet and chisel were used, he had not seen a single case in which the concussion had caused the slightest damage. When a search of the literature showed only six cases in which the result could be attributed to the use of the mallet and chisel, he was inclined to think that there was very little danger of concussion in these cases.

Dr. RAE inquired whether he was correct in understanding Dr. Kopetzky to say that he considered post-auricular oedema to be a symptom of involvement of the sinus. With regard to the first case reported, he would like to know whether the sinus was operated on in a case brought in from the clinic. The lack of temperature symptoms afterward does not indicate that they may not have been present before the opportunity offered to have accurate temperature records taken.

Dr. KOPETZKY, replying to Dr. Rae's inquiry in regard to post-auricular oedema, said that he had noticed that where cases come under observation and there is a swelling behind and above the ear presenting the clinical aspect of a subperiosteal abscess, and there is nothing but an infiltrate found upon incision, he finds that the sinus is usually involved. He was not prepared to say that the sinus contained a clot, but in the majority of such cases he finds either a perisinal abscess or pus somewhere about the sinus. This is in line with the observations which he published last year in the *Annals of Otology*. The first case had been under observation and had been seen by him three times a week since October, and the patient had distinctly said that he had no chills, no fever, and had passed a comfortable night; and it was difficult to convince the patient that he needed an operation.

Dr. GRUENING said that the symptom of oedema of the mastoid in sinus thrombosis is well known as the Griesinger symptom.

Case of sinus thrombosis. Presented by Dr. A. P. VOISLAWSKY.

In this case the temperature continued after the operation on the sinus. This patient has been in Dr. Berens's clinic for two years, and came in with an acute exacerbation on top of the chronic condition. A radical operation was performed, and a sinus discovered the size of a ten-cent piece. There were some granulations, but after uncovering as far as was thought safe the patient was put to bed. The temperature continued, however, and on the 16th he had a chill and a second operation had to be performed. The sinus was exposed half way back to the torcular and was found to have collapsed, while there was considerable bleeding from the emissary vein when exposed. Gauze was applied at each end of the sinus and an incision was made. For a moment there was no return flow of blood, but the operator was unable to get a clot. The blood finally returned both ways. Following the second operation, the patient's temperature continued up and down for the next ten days and he had several chills. Another interesting point was that on September 17th his leucocytosis was 30,000, polynuclears 88 per cent. From that time on this continued down, and in 10 days there was a decrease from 30,000 to 16,000, and the polynuclears dropped to 72 per cent. The patient made an uneventful recovery.

Dr. BERENS said that the remarks made by the Secretary about a drop in the temperature immediately after the removal of a septic thrombus does not always hold good—in fact, it has been his experience that in many of these cases there is a phlebitis accompanying the thrombosis in the lateral sinus and extending beyond the thrombus, besides the general constitutional involvement. Until these clear up, there is apt to be an irregular high temperature. This was illustrated very clearly by Dr. Voislavsky's case. Dr. Berens has seen cases post-mortem in which the phlebitis extended from the jugular into the innominate vein.

Dr. GRUENING said that in the Mount Sinai Hospital service they had frequently noticed high temperature after the removal of a clot, and the culture of the blood showed that they were dealing with a streptococæmia; and the

temperature became normal when that ceased. Only when the blood became sterile did the temperature become low.

Dr. DENCH said that four days before he had operated upon a case which presented some unusual findings at the time of operation. The patient was a woman, twenty-four years of age, who had suffered for thirteen years from suppuration from the right ear. She had been under local treatment for several weeks before she was seen by the speaker. When first examined by the speaker there was a history of vertigo for one week, with some vomiting. There had also been severe pain in the head for about a week. On examination, the right ear presented the characteristic appearance of chronic suppuration, with the presence of granulation tissue. Following out the dictum which Doctor Dench made at the Otological Society last year, the granulation tissue was not removed, and the patient was kept under observation for several days. Further examination showed that there was no nystagmus, and that there was slight tendency to fall toward the affected side when the patient walked with the eyes closed, or when she jumped forward or backward with the eyes closed. Rotation caused no increase in vertigo. On operation a moderate-sized cholesteatoma was found, filling the mastoid antrum. On exposing the horizontal semicircular canal, a fistula was found, of about one-quarter inch in length, and a smaller fistula was also found in the superior semicircular canal. The roof of the antrum was wanting, and the dura was covered with granulation tissue. The foot-plate of the stapes was found in position. The openings in the semicircular canals were enlarged, and the vestibule freely drained posteriorly. Owing to the fact that the foot-plate of the stapes was present, the oval window was not opened, and the cochlea was not disturbed. Upon recovering from the anæsthetic, the patient had no vertigo, and there was no nystagmus. After the first three days there was a profuse discharge of cerebro-spinal fluid, which necessitated reinforcement of the dressings three or four times in each twenty-four hours. The discharge of cerebro-spinal fluid was gradually diminishing. There had been no nystagmus until on the fourth day after the operation, when there was slight nystagmus on looking

to the opposite side. From the profuse discharge of cerebrospinal fluid, which was turbid at the time of operation, there seems to be no doubt that the patient was suffering from a serous meningitis when operated upon. Owing to the extensive destruction of both the horizontal and superior semicircular canals, it is quite surprising there was no nystagmus, and that the vertigo was not more severe. The patient is making an uninterrupted recovery.

Paper: The application of Arneth's blood charts in the diagnosis of obscure mastoid conditions. By ROBERT MILLIGAN, M.D. (Pittsburg).

Discussion.—Dr. SONDERN said that he had been very much interested in the paper read by Dr. Milligan; that any additional light to be obtained from the blood examination of value in diagnosis is indeed welcome.

The use to which Dr. Milligan puts the Arneth count is certainly original and different from what Arneth and others describe. Milligan uses the increase in the relative number of 1-nuclear polynuclear cells as a diagnostic factor in suppurative inflammation, while Arneth seeks only prognostic value in the number of true phagocytes present. Arneth's basal theory is, that the 1- and 2-nuclear polynuclear cells have no phagocytic power while the 3-, 4-, and 5-nuclear polynuclear cells are the true phagocytes. With a knowledge of the total leucocyte count and the polynuclear percentage, he obtains the number of polynuclear cells in *1 cmm* of blood. The actual number of 3-, 4- and 5-nuclear polynuclear cells in *1 cmm* of blood is the basis for his "Index of Phagocytic Capacity." For example, if there are 30,000 leucocytes and 80% or 24,000 polynuclear cells, and of the latter 3% are 3-nuclear, 2% 4-nuclear, and 10% 5-nuclear, we have 5% of phagocytic cells, or an Index of Phagocytic Capacity of 1200.

Another noteworthy point is the difference in the normal figures quoted by Arneth and those by other observers. Arneth's normal figures are 4 to 9% 1-nuclear, 21 to 47% 2-nuclear, 33 to 48% 3-nuclear, 9 to 23% 4-nuclear, and 2 to 4% 5-nuclear, with an Index of Phagocytic Capacity of 2000. Kownatski (*Beitr. z. Geb. u. Gyn.*, x., 1906) makes his

normal figures in the same order—24, 45, 25, 5, and 1. The figures obtained by Smith and Lansing (*Lying-in Hosp. Bulletin*, March, 1908) are practically the same as those published by Kownatski, namely, 24, 39, 28, 8, 7, 0.3. These differences can only be explained by different conceptions of what are 1-nuclear and what are 2-nuclear cells. The count is certainly not an easy matter and great care must be taken if the results of one observer are to be compared with those of another.

A number of favorable results with the Arneth count have been reported, among others by Chace, in the *Post-Graduate Journal*. He calls attention to the fact, however, that it is of no use in chronic cases.

On the other hand, others have attacked Arneth's basal theory. For example, Kaplan, in the *N.Y. Med. Journal*, April 13, 1907, denies that the 3-, 4- and 5-nuclear polynuclear cells have the greatest phagocytic power. He used staphylococci and defibrinated blood, and found that the 1-nuclear polynuclear cells had the greatest phagocytic capacity. Smith and Lansing, in the article quoted, described their examinations of quite a number of smears obtained from cases of gonorrhœal ophthalmia. They found the polynuclear cells containing the largest number of organisms in the following order, 2-nuclear, 1-nuclear, 3-nuclear, 4-nuclear, and that the 5-nuclear cells contained the smallest number. This experimental work would tend to overthrow Arneth's original claim, and the figures I am about to cite from the article by Smith and Lansing would seem to indicate that the Arneth count, as originally proposed, is of doubtful value clinically considered.

These observers made 315 counts in 138 cases. The average in 30 cases of normal pregnancy was as follows: 48.9% 1-nuclear, 36.5% 2-nuclear, 12.6% 3-nuclear, 1.9% 4-nuclear, 0.1% 5-nuclear, I. P. C. 1152. The average in 61 cases of a febrile parturition was as follows: 59.6% 1-nuclear, 29.7% 2-nuclear, 9.2% 3-nuclear, 1.4% 4-nuclear, 0.1% 5-nuclear, I. P. C. 692. In 156 cases of inflammatory lesion without known suppuration, the average was as follows: 64.4% 1-nuclear, 27.3% 2-nuclear, 7.35% 3-nuclear, 0.7% 4-nuclear, 0.05% 5-nuclear, I. P. C. 840. In 34 cases of inflammation

with suppuration the average was as follows: 69.8% 1-nuclear, 25.6% 2-nuclear, 4.2% 3-nuclear, 0.4% 4-nuclear, 0.02% 5-nuclear, I. P. C. 401.

Counts were made in 29 fatal cases. The two extremes were as follows: 12, 69, 10, 5, 4, I. P. C. 984. The other 97, 3, 0, 0, 0, I. P. C. 0. The average was as follows: 74.7, 20.2, 4.5, 0.3, 0.1, I. P. C. 963. The extreme variations in the above figures indicate how unsatisfactory they are to the clinician.

Aside from the marked variation, if we look at the figures from Dr. Milligan's point of view, they seem quite interesting. It must be remembered that these figures were obtained in cases of pregnancy, while Dr. Milligan's figures are from cases of bone lesion, suppurative in character, chiefly in young children. In this connection it is well to recall that suppurative processes in bone do not give the pronounced changes in the blood noted in similar lesions in the soft parts.

In conclusion Dr. Milligan is to be congratulated on the brilliant results he has obtained from his original manner of using the Arneht count, and every effort should be made by other observers to corroborate them.

BOOK REVIEWS.

V.—**A Text-Book of the Diseases of the Ear.** By MACLEOD YEARSLEY, F.R.C.S., Senior Surgeon to the Royal Ear Hospital. 452 pages. London: Kegan Paul, Trench, Trübner & Co., Ltd. 1908. Price 18s., net.

This book is an elaboration of the author's "Common Diseases of the Ear." It is practical, concise, and up-to-date. The arrangement of the subject matter is the usual one; there is added an excellent chapter on the influence of general diseases on the ear. The illustrations are generally new and adequate; literary references are furnished on the most important topics. The get-up of the book is the usual admirable one of the English publishers. Mr. Yearsley has written a very useful text-book and one which can be recommended.

A. K.

VI.—**The Surgery of the Ear.** By Dr. SAMUEL J. KOPETZKY. Illustrated. 368 pages. New York: Rebman Co., 1123 Broadway. 1908. Price \$4.00, net.

This is the fourth American book on the surgery of the ear which has appeared during the last few years. Though the subject has not advanced to this extent, this book differs from its predecessors in that it aims at completeness. The subject is divided into: operations on the external auditory canal, paracentesis, aural polyps, operations to improve hearing in dry middle ear lesions, ossiculectomy, the simple mastoid operation, the radical mastoid operation, the surgery of the labyrinth, operations on the blood-vessels, the surgery of the meninges, surgery of brain abscess, paralysis of the facial nerve, lumbar puncture, ventricular puncture. Each chapter treats of the surgical anatomy, indications, technique, and after-treatment. The author has made a painstaking

study of the German literature and one of the main features of this book is the collection and translation of the opinion of the leading German authorities which should prove very useful for those unable to read German or to whom these references are inaccessible. The illustrations are good and the description of the operations is lucid. The book will prove of interest and useful to those occupied in this field of surgery.

A. K.

VII.—**Lehrbuch der Ohrenheilkunde für praktische Aerzte und Studierende (Text-Book of Otology).** By Professor A. POLITZER. Vienna. Fifth revised edition. 693 pages. Stuttgart: F. Enke, 1908.

Seven years have passed since the appearance of the fourth edition. The history of Otology has only just been completed, and Politzer with that remarkable working power has completely revised and brought out another edition of his excellent text-book. The main changes will be found in the chapters on the physiology and pathology of the internal ear, especially the terminal distribution of the auditory nerve, nystagmus, functional examination of the vestibular apparatus, and labyrinthine suppurations.

Typical otosclerosis according to Politzer is a clinically well-defined disease. "Spongification of the labyrinth-capsule" is not typical for otosclerosis as this condition may occur in the adhesive middle-ear processes. As for the congestive treatment, the author states that Bier's congestive bandage recently recommended as pain-relieving has not proved successful in his experience. The same holds good for the recently advocated suction apparatus of Clapp. Among other new features are radiographs of the ear, operations on the jugular bulb, the most recent plastic methods in the radical operation and intra-auricular transplantations. The new edition will doubtless be received with the same favor, and the text-book will continue to occupy the leading position.

G. BRÜHL (Berlin).

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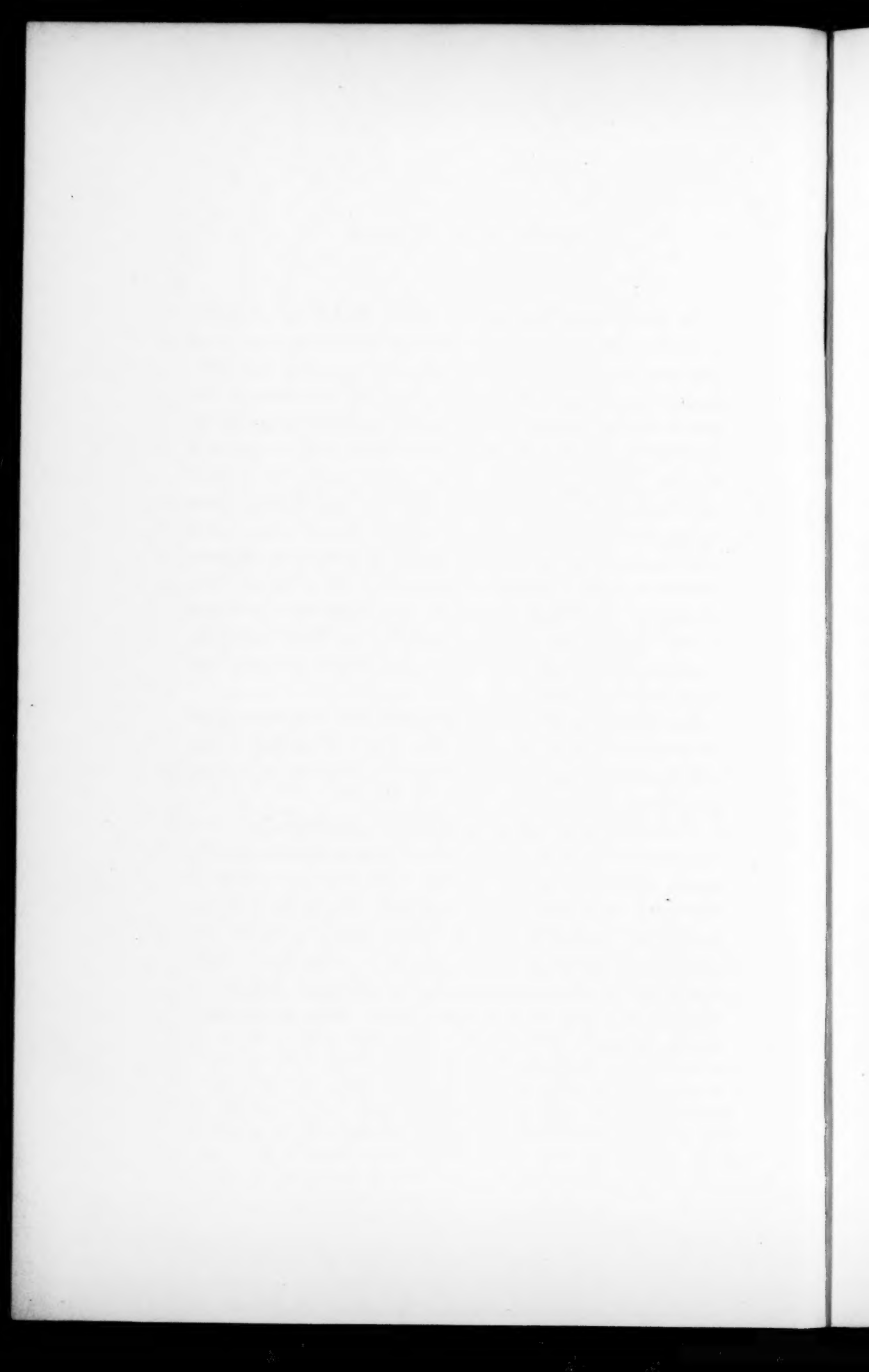
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